LANE MEDICAL LIBRARY STANFORD
L341 .K27 1927 OLD
Nerve tracts of the brain and cord : ana
24503216207

LANE
MEDICAL

LIBRARY

Gift.
University of Texas. Board of
Regents.

AMERICAN BANK NOTE CO. LITHO

Digitized by the Internet Archive
in 2025

https://archive.org/details/nervetractsofbra0000will

NERVE TRACTS OF THE BRAIN AND CORD

THE MACMILLAN COMPANY
NEW YORK · BOSTON · CHICAGO · DALLAS
ATLANTA · SAN FRANCISCO

MACMILLAN & CO., LIMITED
LONDON · BOMBAY · CALCUTTA
MELBOURNE

THE MACMILLAN CO. OF CANADA, LTD.
TORONTO

NERVE TRACTS OF THE BRAIN AND CORD

Anatomy :: Physiology :: Applied Neurology

BY

WILLIAM KEILLER, F. R. C. S., ED.

PROFESSOR OF ANATOMY AND APPLIED ANATOMY,
UNIVERSITY OF TEXAS

New York

THE MACMILLAN COMPANY

1927

All rights reserved

LANE LIBRARY

COPYRIGHT, 1927,
BY THE MACMILLAN COMPANY.

Set up and electrotyped.
Published September, 1927.

PRINTED IN THE UNITED STATES OF AMERICA
BY THE CORNWALL PRESS

LAW LIBRARY

1927
1927

DEDICATION

DEDICATED TO MY STUDENTS IN NEUROLOGY OF THE PAST TWENTY YEARS, WHOSE EARNEST WORK AND SYMPATHETIC ATTITUDE HAVE EVER BEEN MY CHIEF INSPIRATION.

WILLIAM KEILLER.

Department of Anatomy
University of Texas
Galveston, Texas
1927

66900

PREFACE

This book on the nerve tracts of the brain and cord is the result of twenty years' experience in teaching the anatomy of the brain and cord in such a manner as to enable students to approach nervous diseases, thinking in terms of anatomy, physiology and pathology, as applied to the nervous system.

Part I supplies a laboratory manual for the study of the nerves and tracts in the central nervous system as they may be demonstrated in the cord and brain stem in the adult and fœtus and in pathological specimens stained for myelin, as well as in early degenerations stained by the Marchi technique.

Parts II and III form the basis of a course of thirty lectures giving, in Part II, a summary of the anatomy and physiology of the nerve tracts, mainly based on newer methods of investigating the autopsy findings in clinical cases, and in Part III, the leading features of the better known nervous diseases, correlating their symptomatology with anatomical, physiological and pathological data.

No effort is made to go into details and only such leading facts are emphasized as should make an appeal to every well-educated physician.

My experience with students encourages me to believe that I have succeeded in presenting the subject in such a way as to furnish a good working scientific basis for an intelligent understanding of the symptomatology and diagnosis of those nervous diseases that come within the domain of the general practitioner.

I am chiefly indebted for my data to Cunningham's Text Book of Anatomy, Dejerine's Semiologie des Affections du Systeme Nerveux, Church and Peterson's Nervous Diseases, Brain, and the Revue Neurologique.

WILLIAM KEILLER.

CONTENTS

PART I

ANATOMY OF THE NERVE TRACTS

PAGE

General Instructions 3

Structure of the Nervous System: The Neuron 5

Phenomena of Degeneration 9

Physiological Classification of the Neurons 12

Efferent Neurons, 12; Pyramidal or Upper Motor Neuron, 12; Lower Motor Neuron, 12; Afferent Neurons, 13; The Lowest Sensory Neuron, 13; Second, Third and Fourth Sensory Neuron, 13; Association Neurons, Spinal, Cerebral and Cerebellar, 13; Linking Together of the Neurons—General View, 14; Sensory Nerves and Tracts—Afferent Paths, 14; Varieties of Somatic Sense and Their Conduction by Peripheral Nerves, 14; Sensory Tracts in the Cord and Brain, 16; General Plan of These in the Cord, 16; Afferent Paths for the Head: Common Sensation, 17; Vestibular Sense, 18; Auditory Sense, 18; Sight, Smell, 18; Motor Tracts in Brain and Cord, 19; Efferent Path from Cerebral Cortex—Pyramidal Tract or Upper Efferent Motor Neuron, 19; Lower Efferent Motor Neuron, 20; The Cerebellar Tonic and Coördinating Mechanism, 21; Cerebellar Afferent Paths, 21; Cerebellar Efferent Paths, 22; Vestibular Equilibratory and Tonic Mechanism, 23; The Striate Body, Neostriatum, Palæostriatum, 23.

Preliminary Examination of Cross Sections of the Spinal Cord and Brain Stem . . . . . .

Spinal Cord, 24; Medulla Oblongata, 28; Pons, 36; Inferior Colliculus, 44; Mesencephalon and Superior Colliculi, 46; Diencephalon, 49; Corpus Striatum, 54.

Microscopic Examination of Cord (Adult) 56

Adult Lumbar Cord, 56; Lower Thoracic Cord, 59; Cervical Enlargement, 60; First Cervical Segment, 61.

Nerve Tracts Studied by Degenerations 62

Early Degeneration as Shown by Osmic Acid Staining, 62; Late Degeneration as Shown by Iron-Hematoxylin or Pal-Weigert Method, 72.

PAGE

Examination of Fetal Cord and Brain Stem 74

Fetal Cord, 74; Medulla Oblongata, 78; Middle of Pons, 85; Upper End of Pons, 91; Forel's and Meynert's Decussations, 94; Mesencephalon (Figure 68), 96.

Short Summary of the Neurons 99

Description of Long Diagram 99

PART II

ANATOMY AND PHYSIOLOGY OF THE NERVE TRACTS

Summary of Anatomy and Physiology 117

Preliminary Note. Comparison of Somatic Efferent and Visceral Efferent Neurons, 117; Lowest Reflex Arc, 118.

The Motor Neuron 119

Lower Motor Neuron in the Cord, 119; Lower Motor Neurons of Cranial Nerves, 120; Higher Control of Lower Motor (Somatic Efferent) Neurons, 124; Higher Control of Lower Splanchnic Efferent Neurons, 125; Physiology of Lower Efferent Neurons, 125; Physiology of Upper Motor Neuron, 127; Function of Upper Motor Neurons, 130; Reflexes and the Upper Motor Neuron, 131; Cutaneous Reflexes, 131; Babinski Phenomenon, 131; Examples of Upper Motor Neuron Lesions, 133; Degenerations Following Upper Motor Neuron Lesions, 134;

The Sensory Neuron 134

General Statement, 134; Preliminary Classification of Modes of Ordinary Conscious Sensations, 135; Sensory Neurons for Pain, Temperature and Hair Sense, 135; Sensory Neurons for Simple Touch, 137; Spinal Paths for Muscle, Tendon, Joint Sense, Etc., in Posterior Columns, 138; Paths for Touch, Pressure and Muscle Sense of Fifth Nerve, 139; Grouping of Second Sensory Neurons in the Mesencephalon and Subthalamic Region, 140; Third and Fourth Sensory Neurons, 140.

Physiology of the Sensory Nerves and Paths 141

Epicritic Sense, Protopathic Sense, Deep Sensibility, 142; General View of Applied Anatomy and Physiology of the Peripheral Sensory Nerves, 143; Experimental Section of Posterior Nerve Roots, 144; Relation to Symptoms of Locomotor Ataxia, 147; Injury to Posterior Root Ganglia, 147; Injury to Posterior Rami of Spinal Nerves, 147; Injury to Anterior Rami of Spinal Nerves, 147; Incomplete Lesions of Nerves, 148; Complete Section of a Peripheral Nerve, 148; Effect of Cord Lesions Involving Sensory Tracts, 149; Lesions of Medial Lemniscus, 150;

PAGE

Lesion of Sensory Neuron for Pain, Heat and Cold, 151; Third Sensory Neuron. Lesions of the Thalamus, 152; Lesions of the Fourth Sensory Neuron (Thalamocortical Neuron) and Lesion of the Sensory Cortex, 153; Upper Afferent Neurons for Visceral Sense, 154.

Sensory Loss in Lesions of Cranial Nerves 154
Vagus and Glossopharyngeal, 154; Chorda Tympani, 156; Cochlear Nerve, 156; Optic Nerve and Tracts, 158; Olfactory Nerve, 162.

Vestibular Apparatus and the Cerebellum, Their Connections and Functions 163

Cerebellar Paths 165

Afferent Tracts to the Inferior Olive 167

Cerebellar Physiology 169

The Corpora Quadrigemina 171

The Extrapyramidal Motor Mechanism 171
Corpora Striata, Nucleus Hypothalamicus, Substantia Nigra, 171.

Fibræ Propriæ of the Cord and Brain Stem 173

Association Areas and Association and Commissural Fibers of the Cerebral Hemisphere 174

PART III

APPLIED NEUROLOGY

Definition of Common Terms 179

The Cord in Its Segmental Relations 183
Plantar Reflex and the Babinski Phenomenon, 184; Bulbocavernosus Reflex, 184; Skin Reflexes, 184; Tendon Reflexes, 185.

General Considerations Bearing on the Interpretation of Symptoms of Nervous Diseases 186
Importance of the Reflex Arc, 186; Irritants and the Upper Motor Neuron, Upper Motor Neuron Convulsions, 187; Lower Motor Neuron Convulsions, 187; Fibrillar Twitchings, 187; Destructive Lesions of Upper Motor Neuron, 187.

Lesions of the Sensory Neurons 188
Paresthesia, 188; Hyperesthesia, 188; Pain in Nervous Lesions, 189

PAGE

Muscle Tone and Decerebrate Rigidity 190

Anatomic and Physiologic Basis of Muscle Tone, 190; Hypertonicity, 191; Posture in Spastic Paralysis, 192; Rigidity in Flexion, 193; Reflexes and Spasticity in Complete Cord Lesions, 193; Contractures, Early and Late, 194; Shock and Diaschisis, 194.

Lesions of the Sensory Nerves and Roots 196

Cutaneous Nerves. Mixed Peripheral Nerves, 196; Incomplete Interruption of Sensory Nerves, 197; Peripheral Neuritis, 197; Ventral Rami of Spinal Nerves, 198; Ganglia of Posterior Spinal Nerve Roots and of 5th, 7th, and 10th Cranial Nerves. Herpes Zoster, 198; Posterior Nerve Roots, 199; Syphilitic Radiculitis, 199; Chronic Diffuse Posterior Radiculitis (Tabes Dorsalis), 199.

Lesions of Sensory Tracts Below the Thalamus 203

Posterior Columns, Primary Posterior Sclerosis, 203; Combined Sclerosis, 205; The Sensory Decussation in the Cord, Syringomyelia, 205; Syringobulbia, 206; Sensory Tracts in the Medulla Oblongata and Pons, 207; Parasympathetic Nuclei in the Bulbo-Pontine Region, 210; Mesencephalic Sensory and Mixed Lesions, 210; Sensory Lesions of the Subthalamic Region, 211.

Lesions of the Thalamus and Sensory Cortex 212

Lesions of the Thalamus, 212; Comparison with Lesions of the Sensory Cortex, 214; Thalamic Syndrome, 215; Sensory Cortex, 217; Localization of Different Forms of Sensation, 218; Sensory Loss in Subcortical Lesions of the Parietal Region, 220; Sensory Loss in Capsular Lesions, 220.

Motor Paralyses 221

Paralysis Due to Muscular Lesions, 221.

Lower Motor Neuron Paralysis 221

Poliomyelitis, 223; Amyotrophic Lateral Sclerosis, 224; Anterior Nerve Roots and Motor Roots of Cranial Nerves, 225; Spinal Nerves, 226; Peripheral Neuritis, 227.

Upper Motor Neuron Paralysis 227

Distribution of the Paralysis, 228; Group Control, 228; Crossed Supply, 229; Skilled Movements Suffer Most, 229; Head and Eyes Escape, 229; Bulbar Paralysis, 230; Cord Lesions, 230; Brown-Sequard Syndrome, 230; Rigidity and Spasticity in Upper Motor Neuron Lesions, 231; Reflexes in Upper Motor Lesions, 231; Concomitant Phenomena, 232.

PAGE

Typical Pyramidal Lesions and Their Special Features 233
Cortical Lesions, 233; Jacksonian Epilepsy, 233; Blocking of Cortical Vessels, 234; Subcortical Lesions, 235; Illustrative Cases, 236; Capsular Paralysis, 237; Coma and Early Flaccidity, 238; Illustrative Cases, 238; Mesencephalic and Pontine Paralyses, 239; Medulla Oblongata, 241; Spastic Diplegia, 241.

Upper Motor Neuron Paralysis Due to Cord Lesions 242
Primary Lateral Sclerosis, 242; Erb's Syphilitic Paraplegia, 244; Combined Sclerosis, 244; Multiple Sclerosis, 245; Friedreich's Ataxia, 245.

Simultaneous Affection of Both Upper and Lower Motor Neurons . 245
Amyotrophic Lateral Sclerosis, 245; Bulbar Paralysis, 245; Transverse Lesions of the Spinal Cord, Complete and Incomplete, 245; Reflex and Autonomic Centers in the Cord, 246; The Bladder and Rectum in Transverse Cord Lesions, 247; Cord Reflex Centers, 248; Symptoms Common to All Transverse Lesions of the Cord, Complete and Incomplete, 248; Examples of Complete Cord Lesions, 249; Partial Cord Lesions, 253; Lesion of One Lateral Half of the Cord, Brown-Sequard Paralysis, 255; Summary of Brown-Sequard Syndrome, 258; Association of Root Symptoms, 259; Level of the Lesion Relative to the Spinal Column, 259.

The Extrapyramidal Infractorical Motor Mechanism 261
The Cerebellum, 261; Its Functions, 261; Symptomatology of Cerebellar Lesions, 263; Analysis of Cerebellar Ataxia, 266; Further Discussion of Cerebellar Symptoms, 268; The Basal Ganglia, 271; Progressive Lenticular Degeneration, 272; Paralysis Agitans, 273.

The Cerebral Cortex as a Whole 274
Functions of the Cortex, 274; General Discussion of Lesions of Cortex, 275; Aphasia, 279; Lesions of the Right Hemisphere, 281; Apraxia, 283; Clinical Cases of Apraxia, 284; Aphasia, a Form of Apraxia, 286; Head's Classification of Aphasia, 287; Decerebrate Rigidity in Man, 290.

SUPPLEMENT

Classification of Common Nervous Diseases 292
Diseases of the Motor Neurons, 292; Lesions of the Sensory Nerves or Tracts, 303; Cerebellum, 315; Corpora Striata and Red Nuclei, 321; Mixed Lesions of Motor and Sensory Tracts, 322; Lesions of the Cortex of the Hemphispheres and of Association and Commissural Fibers, 323.

Tables of Segmental Supply of Muscles 330

Illustrations 333

Index 435

PART I

ANATOMY OF THE NERVE TRACTS

NERVE TRACTS OF THE BRAIN AND CORD

PART I

ANATOMY OF THE NERVE TRACTS

Internal anatomy of the brain and cord. The internal anatomy of the brain and cord can be learned only by the study of macroscopic and microscopic sections. From naked eye sections of hardened specimens the general gross relations may be learned. This knowledge is to be supplemented by the study of microscopic sections treated by various stains, by a comparison of sections of adult and embryonic brains, and by further comparison of these with the tract degenerations in animals subjected to experiment and with the degenerations following various organic diseases in man. By these methods much may be learned of nerve centers and tracts and their functions.

For purposes of study neither brain nor cord should be cut without preliminary hardening. Something may be learned by immediate section of fresh specimens, but the sections immediately lose all form and become unsuitable for microscopic study. It is always better in nervous cases to embalm the entire body with 10% formalin twenty-four hours before the autopsy. If this cannot be done, the brain and cord should be removed, keeping the vessels long, and the brain should be injected with 10% formalin and 1% sodium chloride through one vertebral and one internal carotid artery, or preferably through both internal carotids as well as one vertebral. The specimens should then be immersed in 10% formalin for at least twenty-four to forty-eight hours before section. In the case of the cord the dura should be opened from one end to the other and the cord (but not the dural sheath) cut across in several places. This prevents distortion of the cord by bending it in the container. Abundant fluid should be used and it should be changed on the first and third days. For further procedure the student should consult Hardesty's Neurological Technique or Mallory and Wright's Pathological Technique or Roussy and l'Hermitte's "Le Technique du Systeme Nerveux." For naked eye study, sections are

cut with a thin, spatula-like brain knife; if this be not available, a long bread knife very sharp will do very well. From cord to mesencephalon inclusive, sections transverse to the long axis alone are needed, but above this level both vertical and horizontal sections are necessary.

If only one brain be available, the mesencephalon should be cut transversely at the level of the separation between the superior and inferior colliculi, and the posterior part used for a series of transverse sections. The anterior part may then be divided in two parts by a section in the line of the great longitudinal fissure and one-half used for horizontal sections while the other half is used for vertical sections. The sections are to be made with pia-arachnoid in place.

The under surfaces of the transverse sections through oblongata, pons, and mesencephalon are to be examined, placed with the anterior aspect toward the observer. Of the vertical sections through the cerebrum the posterior surfaces are to be examined; and of the horizontal sections the superior surfaces are to be studied with the posterior end toward the observer. The most useful sections are: 1. Transverse sections through the cord at the level of the middle of the conus medullaris, the middle of the lumbar enlargement, the first lumbar segment, the mid-thoracic region, the middle of the cervical enlargement, and the 2nd cervical nerve. 2. Transverse sections through the oblongata at the level of the middle of the pyramidal decussation, the greatest swelling of the clava, the cuneate tubercle, the tuberculum cinereum, and middle of the olive, and the upper border of the medulla oblongata. 3. Transverse sections through the middle and upper border of the pons, the middle of the inferior colliculus and the middle of the superior colliculus.

Where possible the sections of the cerebrum are to be retained in their normal relations. Of these the most useful horizontal sections are one through the middle of the genu and splenium of the corpus callosum, one just beneath the body of the fornix, one through the middle of the massa intermedia, and one through the anterior commissure.

Of possible transverse sections the most useful are one through the genu of the corpus callosum, one just in front of the anterior commissure, one through the formamen interventriculare, one through splenium of the corpus callosum, and one section one inch behind this. When possible the whole brain should be cut into serial sections 0.5 cm. in thickness both horizontal and transverse. The brain stem should be included in the transverse sections.

In all sections of cerebrum and cerebellum, whether fresh or hardened

in formalin, cortical gray matter and the gray matter of the basal ganglia are dark in comparison with the white matter. This contrast is emphasized by hardening in chrome salts. In the cord and oblongata however the gray matter is darker than the white matter in fresh specimens only. Even after twenty-four hours hardening in 10% formalin, and still more after hardening in chrome salts, the white matter of the cord is decidedly darker than the central gray matter (so long as the section passes *across* the general direction of the fibers), but in the oblongata, pons, and cerebellum in proportion as the sections cut the white fibers in the direction of their course to that extent is white matter paler than gray matter. Also in the cerebrum, where white bands or laminæ are cut across the direction of the fibers, the color is darker, more nearly approaching the gray matter in shade. Hardening in Orth's fluid is a very valuable method of making degenerative changes in the spinal cord apparent to the naked eye. The author has found the naked eye appearance of sections of unhardened cords exceedingly deceptive.

As the study of the sectional anatomy of the brain and cord is undertaken mainly that one may understand brain physiology and later, nervous diseases, it is better here to summarize briefly some important facts regarding the elements of which the nervous system is constructed.

GENERAL STRUCTURE OF THE NERVOUS SYSTEM

The nerve cell. Of the volumes of information published about nerve cells a few facts must be kept prominently in mind that one may study the nerve tracts intelligently. The whole nervous system is made up of neurons imbedded in a neuroglia network, and is supplied by vessels each carrying into the nerve mass a delicate pial connective tissue sheath with a surrounding lymph space (the Virchow-Robin space).

The neuroglia consists of small stellate cell bodies almost all nucleoplasm; each cell is the source of a dense fine network of processes seen by the use of special stains. The neuroglia, like the neurons, is of epiblastic origin. It has probably a supporting and not a nervous function, and it increases markedly in all inflammatory and degenerative processes, replacing neuron elements. Normally it forms a comparatively thick layer under the pia and round the central canal of the cord and in the walls of the ventricles. The epithelial lining of the central canal of the cord, and the ependymal lining of the ventricles are formed by neuro-epithelium which has undergone only slight modification from its embryonic condition. For our purpose neuroglia need not concern us further.

The term *neuron* means a nerve cell and all its processes, and it cannot be too strongly emphasized that cell body, dendrites, and axoms are one continuous entity, every part depending for its life on the integrity of the whole. The myelin sheath and neurilemma are not essential constituents of the neuron, as the neuraxons in the central nervous system have no neurilemma (primitive sheath) and the gray axoms of sympathetic neurons have no myelin. Still the myelin sheath rapidly degenerates where an axon is cut off from the parent cell.

Neurons appear very differently according to the staining agent used. Figure 29 chosen as a type is a schematic figure of a lower motor neuron, that is, a multipolar nerve cell of the ventral gray column of the spinal cord and its processes. The cell and the first part of its axon are shown as they would appear stained by Nissl's technique with methylene blue, the myelinated portion of the axon is shown as it would appear stained by osmic acid or Weigert's hæmatoxylin, and the telodendria (or terminal arborizations of the axon) are shown as stained by methylene blue.

The *cell body* has a nucleus near the center of the cell; its cell substance is rich in material with a strong affinity for methylene blue when treated according to a definite technique. This material is called tigroid substance from the spotted appearance it gives the cell; it increases with rest, decreases with fatigue and under the action of poisons and disease, and disappears as the cells degenerate.

The cell possesses many branching *dendrites* whose function is receptive. These arise as thick outgrowths containing prolongations of the tigroid substance. They branch rapidly and with great complexity, getting finer as they divide. (Compare the dendrites of Figs. 30, 31, 32 and 33.) Often they have buds or varicosities (Fig. 33). They are never clothed in myelin nor neurilemma. They come into contact relation with the telodendria (branched endings) of the axons of other cells (Figs. 72, 71 and 74) and thus they receive nervous stimuli from these which they in turn transmit to the cell body and thence by the axon or transmitting process to muscles, to other nerve cells or to secreting cells.

The *axon* (Fig. 29) is the transmitting process. It springs as a single process from the cell by a conical projection called the axon hillock, which is devoid of tigroid substance. At first the axon is naked, having no myelin nor neurilemma. Most axons rapidly acquire these but there are some exceptions. The olfactory nerve has a primitive sheath (neurilemma) but no myelin. The optic nerve has myelin as far as the lamina cribrosa where it loses the myelin sheath, but like the nerve fibers in the white substance

of the brain of which the optic nerve is really a direct prolongation, it has no neurilemma. Sympathetic nerves (gray rami) have neurilemma but no myelin.

Nitrate of silver stains show that the number of unmyelinated fibers both in peripheral nerves and in the central nervous system is very large. Probably all the peripheral nerves conveying skin pain, heat, and cold are unmyelinated. These enter the posterolateral column of the cord which chiefly consists of unmyelinated fibers. Even the pyramidal tracts are largely mixed with unmyelinated fibers of cortical origin. The globus pallidus, putamen, and caudate nucleus, and the medial nucleus of the thalamus as well as the gray matter of the cord and brain stem are all enormously rich in non-myelinated fibers. All ultimate synaptic networks are non-myelinated, and the complete neural network of Golgi type II cells (Fig. 33) is also unmyelinated.

The naked axon (Fig. 29) may give off side fibrils in the gray matter (Figs. 30 and 32); the myelinated axon gives off collaterals but does not branch complexly like dendrites, nor does the axon diminish in thickness as it branches. Where there is a neurilemma the collaterals are given off at a node of Ranvier (Fig. 29). Each axon ends in one or more end-arborizations or telodendria; its sheaths are lost just before the terminal branching. In the typical motor neuron under consideration the axon after branching loses its sheaths just as it enters a muscle fiber and in this fiber forms a much branched process with many varicosities called a motor end organ.

All neurons with long axons which leave the gray substance of the brain or cord are called Golgi type I cells. Figures 30, 31 and 32 show examples of Golgi type I cells from the cerebral cortex, from the anterior gray column of the cord, and from the cerebellar cortex, as they appear when stained with silver nitrate by the Golgi process. This method precipitates silver in the cell body, dendrites, and axon, all of which appear solid black. Ramon y Cajal has spent many years investigating the characters of neurons by this method and his figures give an idea of the marvelous complexity of the possible interconnections of nerve cells. Figure 30 is a pyramidal cell from the cerebral cortex (Fig. 27). Its many dendrites, their arrangement in this type of cell, the gemmules with which they are covered, the delicate axon with a side fibril, are well shown. Such an *axon if it belong to a giant cell in the leg area of the anterior central convolution* may extend as one continuous process to the lumbar enlargement of the cord. As it passes from the parent cell it gives collaterals to the corpus callosum which connect it physologically with similar cells in the leg area

of the opposite hemisphere. In the pons it gives collaterals to the pontine nuclei, and in the lumbar enlargement of the cord it becomes connected by its terminal and collateral telodendria with the dendrites of many motor cells (lower motor neurons) either directly or through intercalated cells.

Figure 31 is such a lower motor neuron from the ventral gray column (anterior horn) of the spinal cord of a human fœtus, stained by the Golgi method, which brings out the complexity of its dendrites and the single delicate axon with a side fibril. Figure 32 is a Purkinje cell from the cerebellar cortex. The number and complexity of its dendrites give a marvelous picture of its receptive possibilities. Its axon shows a side fibril passing back into the cortex cerebelli in the neighborhood. The axon soon acquires a myelin sheath and terminates by telodendria which form contact with cells of the dentate nucleus. Such contact between the telodendria of an axon and the cell body or dendrites of another neuron is called a "synapse."

Peripheral sensory or centripetal neurons (afferent neurons) are also Golgi type I cells but they differ a good deal from the type I motor or centrifugal neurons just described. They are illustrated by Figs. 43 to 49. The cell body of each peripheral or sensory afferent nerve is situated in a ganglion of a posterior spinal nerve root or corresponding ganglion of a sensory cranial nerve. Each cell body in Figure 43 has only one process in the adult condition. The smaller cells in Figure 43 give rise to processes which do not acquire a myelin sheath but soon acquire a neurilemma. These smaller neurons probably convey pain, heat, and cold.

Each large cell gives rise to a process which acquires a myelin sheath almost at once and soon divides into one peripheral and one central process, each myelinated. The peripheral processes of the various cells pass to skin, muscle, tendon, joint, or bone, or in the case of splanchnic afferent nerves to glands, involuntary muscle, mucosa, or serous membrane, where they end in receptive end organs. The receptive end organs according to their character and location are capable of receiving impressions of touch, pain, heat, cold, or pressure, or of muscle, tendon, joint, or bone sense, or of visceral sense. Several types of end organs are illustrated in Figs. 45 to 49 as they appear when stained by methylene blue in living or recently dead tissues. The type of sensation transmitted depends on the character and location of the end organ, not on any difference in the nerve fiber. These organs may be far removed from the cell, as are the touch corpuscles in the fingers from the cell ganglia in the posterior nerve roots of the corresponding segments of the spinal cord; but physiologically the periph-

eral process is a dendrite specialized. It is unnecessary at present to speak of the special nerves of smell and sight where the type is somewhat obscured. Examination of Figures 45 to 49, showing receptive end organs of different types makes one think of dendrites far removed from the parent cell to meet special conditions. The central transmitting processes of these sensory neurons, myelinated or unmyelinated as the case may be, soon enter the spinal cord thus forming the posterior nerve roots, or they enter the brain stem to form the sensory roots of the cranial nerves. Each invariably divides into an ascending and a descending branch, each of which sends many collaterals to cells in the neighboring dorsal and the ventral gray columns or corresponding cell groups (nuclei) in the brain stem. (Figs. 44, 71, 72 and 74.) The posterior nerve roots which convey muscle sense soon cease giving off collaterals and ascend in the dorsal white column to end by telodendria round the cells of the nucleus gracilis or nucleus cuneatus (Fig. 73). Shorter nerve roots carrying impressions of muscle sense for the cerebellum or of touch, pain, heat, or cold, ascend only a short distance in the cord before ending round varying groups of cells in the dorsal gray column whence they are relayed as will be seen later (Fig. 74).

Neurons whose function is to connect different regions of the cord and whose axons form the fasciculi proprii of the cord (see Fig. 53 and Fig. 74) furnish another example of Golgi type I cell. In Figure 74 each of these cells is seen to have many dendrites which form synapses with the telodendria of the axons or collaterals of other cells. (Compare Fig. 72.) The axon may divide into an ascending and a descending branch (Fig. 74) each of which in turn sends many collaterals into the gray matter to connect and coördinate the action of many motor neurons.

The Golgi type II cells, illustrated by Figure 33 from the cerebral cortex, differ from the type I cells in that the axons of this type never leave the gray matter, and though complex, never go a great distance. They link together cells of type I in neighboring areas of gray matter. The axon of this type of cell is much branched, giving off many side fibrils. It is delicate and differs markedly from the dendrites. These Golgi type II cells are very widely distributed in the cortical gray matter of the cerebrum and cerebellum, in the basal ganglia and optic thalamus, and in the gray matter of the brain stem and cord. All the nerve cells of the retina except the rods and cones and the cells of the ganglionic layer are Golgi type II cells.

Phenomena of degeneration. So complex are the relations of the neurons that histological preparations alone throw but little light on the

more distant connections of the processes. Fortunately much information may be gained by the study of the leading phenomena of degeneration in axons and cell bodies. Figs. 35 to 42 are especially designed to illustrate these.

Wallerian degeneration. When an axon is cut off from its connection with its cell body it dies, the separated portion undergoing a rapid degenerative process which progresses with equal rapidity in its whole length. The portion of the axon still attached to the cell does not degenerate in the same rapid way. Figure 37 illustrates this. In Figure 37a, a normal nerve is shown, the myelin stained black with osmic acid. In Figure 37, b and c, two different stages of degeneration are seen. The myelin no longer forms a continuous sheath between the nodes of Ranvier with the axis cylinder in the middle, but is reduced to droplets of fat irregularly distributed. Figure 38 shows a normal nerve in transverse section fixed by the usual method and stained with osmic acid. The regular circles of cut myelin sheaths are stained black by the acid. Now when a segment of nerve or spinal cord is hardened in Muller's fluid containing chrome salts, normal myelin does not stain with osmic acid but degenerated myelin does so stain because of the fat droplets present. This black staining appears about two weeks after the lesion and persists for two and a half months or more. After this time the fatty substance into which the myelin has degenerated disappears entirely. The technique is known as Marchi's, and Figure 39 shows a small portion of a section of spinal cord with degenerated fibers hardened and stained by this method. The normal white substance is lightly stained, a dirty yellow. The degenerated fibers are indicated by irregular droplets of fatty matter stained black, not by circles as normal myelin would be stained. Low power pictures of the same degenerated tracts are seen in the Figures 77 to 81. Thus within certain time limits it becomes possible to trace degenerated tracts in the central nervous system after lesions produced by experiment or disease (consult Figs. 77 to 81, and also Figs. 40 and 41.) Figure 77 gives positive pictures of tracts degenerated after experiments in monkeys, and Fig. 78 of tracts degenerated by disease in man, stained by Marchi's method with osmic acid after hardening in Muller's fluid. Figure 39 shows a small portion of such a picture under a high power. Figure 41 is a low power picture of a degenerated tract in one of the sections illustrated in Fig. 82, stained by scarlet red, also a selective fat stain.

Another method of tracing degenerated tracts useful when the degenerated fibers are massed together is illustrated in Figure 82. Here the

degenerated fibers are those that fail to hold the stain under certain bleaching agents. The normal myelin sheath stains dark purplish blue or bluish purple by hæmatoxylin when treated according to the Pal-Weigert or Heidenhain iron-hæmatoxylin technique. The degenerated tracts which have lost their myelin do not hold the stain when the prescribed agent is used. Thus when sufficiently massed together degenerated tracts may be distinguished as unstained areas among darkly stained groups of normal fibers. This method does not depend on the presence of fat droplets and is therefore possible long after the Marchi method has ceased to be available; but as the picture is a negative one, isolated degenerated fibers do not show clearly. On the other hand, the Marchi method demontrates degenerated fibers even when much scattered (see Fig. 77) but is only applicable within very definite time limits, as the fatty droplets are absorbed three months or more after the division of the axon.

Chromatolysis. The portion of the axon attached to the cell body does not show rapid degeneration of its myelin sheath. However when the axon is cut and the cell ceases to be capable of functioning, the tigroid substance of the cell body undergoes degenerative changes as shown in Figure 36. The tigroid substance of the cell rapidly disappears, the cell body fails to stain darkly with methylene blue, toluidin blue or other basic dyes. Later the cell body becomes cloudy and the nucleus is displaced to one side. Very slowly the nerve cell atrophies and completely disappears.

Retrograde degeneration of the axon. Although the part of the axon remaining attached to the cell body in the cord does not show fatty degeneration of its myelin sheath as rapidly as the axon severed from its parent cell, yet if a motor nerve root be forcibly pulled away from the brain stem, the myelin of the proximal part of the divided axons undergoes a slow fatty change. It stains by Marchi's method if the animal be killed thirty-five to forty days after the injury. This method of indirect degeneration has been largely used by van Gehuchten to demonstrate the intramedullary course of motor cranial nerve roots. By the use of such histological and physiological methods the grouping and interrelations of cells and their processes are gradually becoming known.

The terms *nucleus, center, ganglion* are used in neurology to describe collections of nerve cells, and in fact have been applied very loosely.

The large multipolar nerve cells in the anterior gray columns of the cord whose axons form the spinal motor nerve roots may be described as forming the nuclei of origin of these nerve roots. In the spinal cord these cells form nearly continuous columns (Figs. 6 to 9 and 53 to 55). In the brain stem

the nuclei of the cranial motor nerves are short, isolated columns of cells (compare Figs. 12, 13, 14, 59 and 60). These cells and their axons which form the peripheral motor nerves are called the lower motor neurons. The nuclei of origin of the ordinary sensory nerves (as distinguished from the nerves of special sense) are the collections of nerve cells which form the ganglia of the sensory roots of the fifth, seventh, ninth, and tenth cranial nerves and the ganglia on the posterior spinal nerve roots (Figs, 10, 11, 12, 13 and 43). These peripheral sensory nerves are the lowest sensory neurons. Their peripheral processes, as we have seen, end in specialized sense organs. Their central processes or axons enter the cord or brain stem and end sooner or later by synapsing with the dendrites of a second sensory neuron. The collection of cells in connection with which these lowest sensory neurons end are called their terminal nuclei.

Here again in the spinal cord these terminal nuclei from which the second sensory neuraxons spring form continuous columns such as the gelatinous substance and nucleus dorsalis; but in the brain stem the sensory end nuclei are less continuous (compare Figs. 12 and 13). The large collection of efferent nerve cells in the caudate and lentiform nuclei, and the collections of sensory nerve cells in the thalamus form the chief infracortial ganglia of the cerebrum.

PHYSIOLOGICAL CLASSIFICATION OF THE NEURONS

(Read with reference to Figs. 69 and 70)

On the basis of physiological function neurons fall into three great groups: efferent, afferent and association neurons.

Efferent neurons. The pyramidal or upper motor neurons form the great pathway by which voluntary motor impulses pass from the motor area of the cerebral cortex to the lower motor neurons in the brain stem and spinal cord. The cells of origin are the large pyramidal cells, called Betz cells, of the motor cortex in the anterior central gyrus (Figs. 27 and 30). Their axon terminals synapse probably indirectly through intercalated cells with the dendrites of the lower motor neurons (see Figs. 69 and 70).

The lower motor neurons are the nerve cells and roots of the cranial and spinal motor nerves. Their cell bodies are in the nuclei of origin or motor nuclei of spinal nerves (Figs. 6 to 9) and of certain cranial nerves, the third, fourth, fifth, sixth, seventh, ninth, tenth, eleventh, and twelfth (Figs. 12 and 14), and their axons form cranial motor nerve roots and anterior spinal nerve roots. The axons end in special motor nerve endings

in striped muscles or by synapsing with sympathetic neurons for the supply of unstriped muscle or of glands.

Afferent neurons. The lowest sensory neurons have their cell bodies in the ganglia of the posterior nerve roots, in the special ganglia of the sensory roots of the fifth, seventh, ninth, and tenth cranial nerves, or still more specialized ganglia in the nerves of special sense (Fig. 43). For ordinary sensory nerves the peripheral endings form end organs in skin, muscle, tendon, periosteum, or joint capsules. Their central processes or axons enter the cord or brain stem and synapse with the dendrites of *separate groups of nerve cells* for each form of sensation. These cell groups form the *second relay or second sensory neurons* transmitting each form of sensation to the thalamus whence a *third relay* conveys each form to the sensory areas in the cerebral cortex (see Figs. 69, 70 and 71).

Note: It is probably more correct to postulate a short relay (*third sensory neuron*) from the ventro-lateral to the dorso-lateral nucleus of the thalamus and from this *two relays,* one to the medial nucleus of the thalamus where elementary sensations reach consciousness, and produce reactions affecting the sensory state of the individual, and one to the cortex (*thalamo-cortical neuron*) connecting with the cortical mechanism for sensory memories, judgments and cortical reactions. These are not shown in Figure 70.

Association neurons.

(*a*) **Association neurons in the spinal cord, and mechanism for cord reflexes.** The lowest afferent or sensory neuron, on entering the spinal cord or brain stem bifurcates into a descending branch and an ascending branch which may be short or long (Figs. 70 and 74). From these many collaterals arise. These may pass directly to the motor cells of the anterior gray columns thus providing for direct reflexes (Figs. 70 to 76); or collaterals at various levels may arborize round the cell bodies of neurons whose axons form the *fasciculi proprii of the spinal cord* (Figs. 69, 70 and 74). These fasciculi proprii link up sensory neurons with widely spread motor neurons and thus provide the anatomic basis for the more complex cord reflexes, as for example, the scratch reflex in the dog (Fig. 76).

Cerebral association neurons (Fig. 149) comprise short and long fibers passing from one area of the brain to another. Only a very limited area of the cerebral cortex is motor or sensory. The great bulk of the cortex is concerned with the storage of motor and sensory memories, the relation and comparison of these, and the formation of habit reactions whether of response or inhibition, with final voluntary action. All this is provided for by innumerable long and short "association neurons" which link together

many areas of cerebral cortex with each other (Figs. 142, 143 and 149). The cells of these association neurons are pyramidal cells smaller than the Betz cells (Fig. 27).

Cerebellar neurons. The cerebellar mechanism for maintaining muscular tone and coördination consists of a whole system of afferent and efferent neurons which will be considered in detail later (Fig. 121).

Linking together of the neurons.

Before commencing the detailed study of the brain and cord, it is advisable to take a general view of the working mechanism of the nervous system. This may be done with the help of Figs. 69 and 70, altered and amplified from Elliot Smith's figure in Cunningham's Anatomy.

Sensory nerves and tracts. The usual type of mixed nerve, as exemplified by the fifth cranial nerve and by most spinal nerves, carries not only efferent impulses, but also afferent or sensory impulses. These sensory impulses arising from skin and from fasciæ, muscles, tendons, bones, and joints, are of various character and may be classified into epicritic, protopathic and deep sense. (See Fig. 99.) Ordinary sensation is exceedingly complex, and a thorough understanding of its varieties is essential.

Body or somatic sense.

Myelinated cutaneous nerves are capable of carrying to the spinal cord and thence by relays to the cerebral cortex the three distinct varieties of sensation which are comprised under the term *epicritic sense.* These are:

(*a*) **Simple touch,** as distinguished from pressure and from the slightly different sense of touch conveyed by the hairs, is tested by a delicate pencil of absorbent cotton. *Hairy parts must first be shaved.* To test this tactile sense with the finger, the head of a pin, or a match is wrong, as these stimuli bring in pressure sense and are not sufficiently delicate in minor degrees of sensory loss.

(*b*) **The perception of slight degrees of heat and cold,** ranging from body temperature up to 104° F. for heat, and down to 72° F. for cold, should be tested by carefully graded metallic tubes of warm and cool water; teaspoons taken from warm or cool water do very well. Marked degrees of heat and cold are not a test of epicritic sense.

(*c*) **Tactile discrimination and localization** means respectively the ability to tell when one is touched by the two points of a compass simultaneously, and to indicate the point touched. The points of the compass are blunted and the distance at which the two points can be recognized as two is read in millimeters. On the tip of the tongue and on the fingers the normal distance is 1 to 2 mm.; on the hand and foot it is 5 to 30 mm.;

on the arm and leg, 40 to 80 mm. This type of sensation is usually called compass sense.

Other cutaneous nerves, probably unmyelinated, carry protopathic sense. This comprises:

(*a*) *Hairsense or tickling* as felt when hairs are delicately brushed.

(*b*) *Skin pain,* whether it be tested as usual by a pinprick, or by pinching the skin or pulling the hairs.

(*c*) *Cold* below 60° F. and *heat* above 120° F., tested by graded metallic tubes (or a teaspoon taken from hot or cold water).

(*d*) Lastly there is *deep sensibility* which is carried by sensory nerves running deeply in the same bundles with the motor nerves supplying muscles. Deep sensibility comprises:

(1) That complex of *muscle, tendon, and joint sense* which enables one to tell the position of his limbs, as well as pressure, and the form and weight of objects as distinguished by touch. This sense must be tested while the patient's eyes are closed. The examiner bends the digits, wrist, ankle, elbow, knee to various degrees of flexion, abduction, etc., and asks the patient to imitate the position so produced; or the patient may be asked to touch his nose or toe, or place his heel on his opposite knee; or he is required to estimate the varying weights of articles of the same size and form; or attempt to recognize by handling familiar objects, such as a dollar or a dime, a key or a knife. These tests are of value only when the patient's eyes are closed.

(2) *Tuning fork vibration,* commonly spoken of as bone sense, tested by placing a vibrating fork against a bony prominence or a finger or toe nail. Muscles also convey vibratory sense as may be tested by a tuning fork on the abdominal wall.

(3) *Deep pain,* that variety of pain produced by heavy pressure, as distinguished from skin pain.

(4) Pressure localization.

Each of these sensory impulses is felt separately by special end organs; even heat and cold has each its special sense organ. All are grouped together in a single sensory (posterior) nerve root, though they probably travel by separate root fila (Fig. 99). As soon as the fila enter the cord the various senses are distributed to different tracts in the cord, (Fig. 69). On recognition of this redistribution depends much of the diagnosis of nerve tract diseases.

In approaching the exceedingly difficult but all important subject of the nerve tracts it is helpful to keep constantly in mind the basic principle

that no efferent mechanism in the nervous system is capable of action except under the influence of afferent impulses. If all the posterior afferent or sensory nerve roots coming from a limb be cut, the limb is just as much paralyzed for all effective purposes as if the anterior (efferent or motor) nerve roots were cut. No center for efferent impulses can act if it be cut off from afferent impulses. It follows that all centers capable of giving off efferent stimuli for purposes of motion or secretion have underlying and necessary afferent tracts. Injury to these afferent tracts will seriously impair the action of efferent centers, and separation from all afferent paths will render the generation and transmission of efferent impulses impossible. The anatomical basis of this physiological principle is that all efferent neurons or collections of neurons are connected directly or indirectly with the periphery by means of one or several sensory neurons from skin, mucous membrane, or motor apparatus—as muscles, tendons, joints, or bones. This fact must be kept constantly in mind if one is to remember the afferent and efferent paths of the nervous system.

Sensory tracts in the cord and brain. All forms of epicritic, protopathic, and deep sensibility travel together in the sensory nerve trunks and enter the cord by the posterior nerve roots, but as soon as they enter the cord there is a complete rearrangement of the paths by which different forms of sensation travel to the brain. The discussion which follows must be read with constant reference to Figs. 69 and 70. The root fibers conveying *muscle-tendon-joint sense,* that is, the sense that enables one to perform accurate manipulations and to recognize the position of one's limbs without the aid of sight, as well as the root fibers conveying *tuning-fork sense,* and *compass sense* travel up the posterior columns of the same side of the cord as far as the medulla oblongata. The fibers from the leg and abdomen shift in the posterior column nearer the posterior median septum as they travel upward. Above the mid-thoracic region of the cord they have become grouped together so as to form a tract known as the fasciculus gracilis. The long fibers for the upper thorax, arm, and neck are grouped on the dorsal surface of the fasciculus cuneatus. The fasciculus gracilis ends in the nucleus gracilis of the medulla oblongata; the long fibers of the fasciculus cuneatus end in the nucleus cuneatus. In these nuclei a fresh relay is formed. The fibers from this relay immediately cross under the name of internal arcuate fibers to form a new tract in the opposite side of the medulla oblongata called the medial lemniscus. (The fibers which relay compass sense appear to take some other course through the opposite side of the oblongata.) The medial lemniscus ends in the

ventro-lateral nucleus of the thalamus where a fresh relay conveys the sensory impressions to the dorsal part of the lateral thalamic nucleus and thence to the posterior central gyrus by a fourth relay. For the sake of simplicity only one relay is shown in the thalamus in Figure 70.

(*Note:* Head has shown that the posterior roots of the sixth to the twelfth thoracic nerves—and on general considerations we may include with these the third to the fifth thoracic nerves—have no representation in the nucleus gracilis or cuneatus. It is therefore probable that tactile discrimination and postural sense are limited to the limbs where alone they have physiological significance.)

Fibers of the posterior nerve roots which convey *heat, cold,* and *pain* travel upward for slightly varying distances (2 to 6 cm.) in the posterolateral column (Lissauer's) and synapse with cells of the posterior gray column, probably in the gelatinous substance. Here they are relayed by neurons which immediately cross in the gray or in the anterior white commissure and thus pass to the opposite posterior spinothalamic tract (see Fig. 69). By this they are conveyed to the thalamus, whence they are relayed to the cortex. Here then is an important contrast. Fibers conveying muscle sense, compass, and tuning fork sense travel up the same side of the cord, that is, homolaterally, *till they reach the oblongata in which they cross;* whereas the fibers for pain, heat, and cold travel up the same side of the cord for a short distance only and are relayed by fresh fibers which cross *in the cord* by way of the commissures and ascend in the *opposite side of the cord* to the thalamus.

Simple tactile sense and pressure sense after a short homolateral ascent are relayed by neurons whose cells are in the posterior gray columns and whose axons cross in the gray or anterior white commissure to form a tract called the ventral spinothalamic (Fig. 69) which ascends to the thalamus. Usually simple tactile sense and pressure sense travel also homolaterally to the oblongata in the posterior columns, to be relayed by the opposite medial lemniscus.

Afferent paths for the head. Common sensation. The sensory root of the trigeminal nerve carries sense of touch, pain, heat, and cold for the eye, nose, mouth, forehead, and face, as well as bone, and probably muscle sense for the face and masticatory and tongue muscles. Its ganglionic cells are in the semilunar ganglion. It enters the pons and terminates mainly—

(*a*) In the lateral sensory nucleus of the trigeminal nerve.

(*b*) In the spinal tract of the trigeminal nerve which descends as far as the second cervical cord segment and ends in the gelatinous substance.

The lateral sensory nucleus is probably homologous with the gracile and cuneate nuclei and is the nucleus of the first relay for touch, pressure, and muscle sense. From this the relay is probably by way of the opposite medial lemniscus to the ventrolateral nucleus of the thalamus, and thence, by way of the dorsolateral thalamic nucleus to the cerebral cortex. The gelatinous substance is probably the end nucleus for pain, heat and cold, and perhaps for touch. For these varieties of sensation the path from the gelatinous substance is by way of a special tract in the opposite side of the medulla and pons—the trigeminothalamic tract. This lies in the formatio reticularis of the oblongata and pons and is sufficiently isolated to be sometimes involved separately in a destructive lesion giving a definite syndrome. The trigeminothalamic tract joins the medial lemniscus near the thalamus in which it terminates, to be here relayed to the essential thalamic organ and cerebral cortex.

Vestibular sense and auditory sense are conveyed by *the eighth nerve.*

(*a*) **Vestibular sense,** or the sense of the position of the head underlying equilibration, has a special mechanism and will be discussed later.

(*b*) **Auditory sense** has a special receptive organ, the cochlea (Figs. 70 and 102) whose nerve is the cochlear nerve. The ganglion cells are in the spiral ganglion of the cochlea. The nerve ends in the ventral and dorsal cochlear nuclei in the lower end of the pons (Fig. 102). From these nuclei relays partly homolateral but mainly crossed form the lateral lemniscus which goes to the medial geniculate body. From this a second relay goes to the superior temporal convolution. Terminals or collaterals of the lateral lemniscus go also to the inferior colliculus for special muscular reflexes associated with hearing.

Sense of sight. (Figs. 105 and 70.) The organ of sight is extremely specialized. The right visual field is projected on to the left half of each retina. The left half of each retina is connected by the left optic tract with the left lateral geniculate body, left pulvinar, and left superior colliculus. These nuclei are relayed to the left calcarine area of the cerebral cortex for visual impressions. The left half of the field of vision is similarly projected on to the right half of each retina and conveyed to the right ganglion and cortex. The visual center of the retina is bilaterally represented, and visual memories are stored in the left occipital lobe only, in right-handed persons; in left-handed persons they are stored in the right occipital lobe only.

The sense of smell is too unique in all its connections for discussion here; it will be taken up later.

It has been shown that all common sensory impulses are relayed from the thalamus to the cerebral cortex. Probably they pass first to the posterior central gyrus (Figs. 90, 140 and 141). Here short association fibers (Fig. 149) form relays to the motor area in the anterior central gyrus for voluntary motor responses, and to an area farther back in the parietal lobe for storage as memories, and for purposes of comparison and judgment.

Besides this all forms of sensation which carry a sense of feeling-tone, of pleasure, discomfort, or pain, are also relayed from the lateral nucleus of the thalamus to the essential thalamic organ (Head and Holmes, *Lancet*, January, 1912). Here they reach consciousness as touch, pain, warmth, cold, etc., and are associated with a sense of comfort or discomfort. This "essential thalamic organ" is probably the medial thalamic nucleus. It is under cortical control through corticothalamic neurons.

Motor tracts in the brain and cord.

Efferent path from the cerebral cortex. The upper motor neurons together form the pyramidal tract. This is the tract for voluntary motion. *The pyramidal tract* is composed of the axons of the large pyramidal Betz cells (Fig. 27) in the motor area of the anterior central convolution (Figs. 70, 90, 91 and 93). These cells are in intimate relation with afferent paths from the skin and muscles by tracts relayed from the thalamus, and also with receptive (sensory) areas and association fields of the cerebral cortex by means of the cerebral association fibers (Fig. 149).

From the anterior central convolution the pyramidal fibers converge to the knee and anterior two-thirds of the posterior limb of the internal capsule (Fig. 87, c, e) occupy the middle three-fifths of the basis mesencephali (Fig. 87, d), where they send off fibers to the nuclei of the opposite oculomotor and trochlear nerves and pass through the pons forming three-fifths of the longitudinal bundles of the pons (Fig. 64 and 63). Here they send fibers to the motor nuclei of both trigeminal nerves (Fig. 63), especially to that of the opposite side.

At the lower end of the pons they are concentrated (Fig. 63) and give off fibers to the opposite sixth (abducent) nucleus and to both seventh (facial) nerve nuclei, especially to that of the opposite side. In the medulla oblongata they form a very sharply defined tract called the pyramid on each side of the ventral median groove (Figs. 60, 59 and 10), while some aberrant fibers are found scattered in the medial lemniscus destined for distribution to the twelfth nucleus. Still other aberrant fibers lie more laterally and remain on the same side (see Fig. 79). In the medulla oblongata, fibers are given to the somatic efferent nuclei of the glosso-

pharyngeal (ninth) nucleus, to the vagus and accessory (tenth and eleventh), and to the hypoglossal (twelfth) nucleus (Figs. 59 and 14). All these nuclei receive a few homolateral pyramidal fibers.

At the lower end of the oblongata about three-fourths of the pyramid can be seen by the naked eye to cross to a lateral position in the spinal cord, forming the *lateral cerebrospinal tract or fasciculus* (O. T. crossed pyramidal tract, Figs. 10 and 58). The lateral cerebrospinal tract contains a few uncrossed fibers (Fig. 79) for the supply of muscles which act together with those of the opposite side of the body as do the intercostal muscles. The lateral one-fourth of the pyramid in the oblongata continues down the same side of the cord where it forms the *anterior or ventral cerebrospinal fasciculus* (Figs. 57 and 56; O. T. direct pyramidal tract); but this is not destined for homolateral distribution. It crosses fiber by fiber until it is exhausted at about the lower end of the cervical enlargement. Sometimes the whole pyramid crosses at the pyramidal decussation in the oblongata and on the other hand the anterior cerebrospinal fasciculus quite often extends down to the sacral region. The pyramidal fibers carry voluntary motor impulses from the motor area of the cerebral cortex to the lower motor neurons. These impulses do not reach the lower motor neurons directly but through intercalated cells. It is customary, however, to speak of the synapses between the upper and lower motor neurons as though they were direct. This pyramidal tract is the only tract in man by which voluntary motion can be produced. If it be destroyed as by a hemorrhage into the internal capsule, the result is loss of voluntary motion in the opposite side of the body (except the eyes and upper part of the face). This is the ordinary form of paralytic stroke, and the condition produced is called hemiplegia. Examples of lesions of the tract are seen in Figure 79, which shows softening of the motor cortex, and Figure 168a, which shows softening of the internal capsule.

Efferent paths from the cord and brain stem.

The lower motor neurons (Figs. 69 and 70). The cell bodies for the spinal motor nerves are arranged in columns of cells in the anterior gray column (Figs. 6 to 9 and 53 to 55). Their axons form the anterior nerve roots and end peripherally in end organs in voluntary muscles (Fig. 29).

The motor cranial nerves arise in groups of motor cells in the mesencephalon, pons, and medulla oblongata (Figs. 12, 13, 14, 59 to 61, and 68).

There is no other lower or peripheral efferent path from cord or brain stem except this lower motor neuron and all motor influences—voluntary,

reflex, or coördinating—must reach the muscles through this, the final common (efferent) path of Sherrington.

The Cerebellar Tonic and Coördinating Mechanism. (Figs. 69, 70, and 121).

Cerebellar Afferent Paths.

There is another variety of sensory impulses which must be kept in mind in considering the tracts of the nervous system. Certain afferent impulses, chiefly from the muscles, never reach consciousness at all, but must be continually conveyed to the cerebellum in order that that organ may exert its tonic and coördinating influence on motor nerve cells and thus on muscular action. To this end peripheral sensory nerves whose end organs are in muscles, tendons, or joint-capsules enter the spinal cord by the posterior nerve roots, ascend a short distance and arborize round the cells of the *nucleus dorsalis* (Figs. 69, 54, and 7). Hence they are relayed homolaterally by the *posterior spino-cerebellar tract* by way of the restiform body to the vermis of the cerebellum (Figs. 70, 60, 121, 11, 15, and 81). Connections also go to Deiters' nucleus, the nucleus dentatus, the flocculus, the roof nuclei and the cerebellar hemispheres (Fig. 81). Other afferent nerves arborize round cells whose location in the posterior gray column is still a matter of conjecture. These are relayed to the vermis by the *ventral spino-cerebellar tract.* This reaches the cerebellum rather circuitously by way of the brachium conjunctivum (Figs. 121, 81).

The afferent spinocerebellar paths for the arm and neck are still imperfectly known. It has been shown that cells in the cervical cord corresponding fairly closely with the position of the nucleus dorsalis send fibers to the dorsal and ventral spinocerebellar tracts, while André Thomas believes that muscle sense for the arm and neck is relayed in the accessory cuneate nucleus (Fig. 59) to the restiform body. The cerebellar afferent path for the sensory distribution of the *trigeminal nerve* is by a relay from the lateral sensory trigeminal nucleus to the vermis (Fig. 62). Similar afferent paths to the cerebellum from the glossopharyngeal and vagus sensory nuclei probably provide for cerebellar influences over deglutition and phonation.

Auditory and visual afferent paths to the cerebellum reach the vermis from the corpora quadrigemina by cerebellar afferent fibers in the brachia conjunctiva.

The peripheral afferent tract to the *cerebellar hemisphere* is probably by way of deep fibers in the cord derived from cells in the gray columns, which receive collaterals from sensory nerve roots. These ascend to the

inferior olive, whence new fibers reach the opposite cerebellar hemisphere through the restiform body. The posterior spinocerebellar tracts also send collaterals to the cerebellar hemispheres (Fig. 121).

Cerebellar afferent paths from the cortex cerebri. Probably all parts of the cerebral cortex, but certainly the frontal, parietal and temporal convolutions are connected with the cortex of the opposite cerebellar hemisphere by corticopontine nuerons. These pass through the basis pedunculi and arborize round the cells of the homolateral basilar part of the pons, whence a relay passes to the opposite cerebellar hemisphere by way of the brachium pontis (Fig. 121).

Cerebellar Efferent Paths. (Figs. 70 and 121.)

All cerebellar efferent impulses start in the cells of Purkinje (Figs. 32, 121) in the cerebellar cortex. The cortical efferent neurons of the vermis and flocculus end in the homolateral nucleus fastigii and nucleus globosus, whence efferent relays pass to the vestibular nuclei of both sides. Many pass alongside the vestibular nuclei without interruption and join the vestibulospinal tract to synapse with the lower motor nuerons. The cortical efferent neurons of the hemisphere end in the corpus dentatum and nucleus emboliformis. The neurons of these bodies form the brachium conjunctivum (Figs. 70, 60, 62, and 63). The brachium conjunctivum intercrosses with its fellow under cover of the inferior colliculi (Fig. 66) and ends partly in the red nucleus and partly in the thalamus (Figs. 70 and 68).

Through this thalamic connection a cerebellar tonic influence may be exerted on the thalamic nuclei and by a thalamocortical relay on the cerebral cortex.

The rubrospinal tract (Figs. 70, 69, and 67) is one of the cerebellar efferent tracts to the lower motor neurons in the brain stem and spinal cord. The rubrospinal tract starts in the cells of the red nucleus and immediately crosses (Figs. 67 and 70). It descends, occupying a position near the spinal tract of the trigeminal nerve in the pons and medulla oblongata and just in front of the lateral cerebrospinal tract in the cord. Its neurons end by arborizing round the cells of the lower motor neurons in the brain stem and cord. (The vestibular mechanism will be considered apart from the cerebellum.)

It is to be noted that practically all cerebellar connections are homolateral. The cerebellar afferent paths are homolateral and the cerebellar efferent paths undergo a double crossing. In contrast to this, all cerebral connections are crossed.

The Vestibular Equilibratory and Tonic Mechanism.

The vestibular portion of the eighth cranial nerve is the afferent nerve of the semicircular canals, and these are the great peripheral organs of equilibration. The vestibular nerve ends in several groups of cells in the lateral part of the floor of the fourth ventricle in the neighborhood of the area acustica (Figs. 70 and 60). Of these, the connections of only one nucleus, a group of large cells called Deiters' nucleus, call for attention here. Its cells give off axons which form the main bulk of the medial longitudinal fasciculus in the medulla oblongata and brain stem above it, which carries vestibular influences to the motor mechanism for turning the eyes and head. Axons from Deiters' nucleus also form the vestibulospinal tract which descends homolaterally in the anterior part of the anterolateral white column of the cord.

These nuerons synapse directly with the lower motor neurons. These two tracts appear to maintain muscle tone and coördinate body movements with equilibratory impressions received from the semicircular canals. Deiters' nucleus receives afferent fibers from the body musculature by collaterals from the ventral and dorsal spinocerebellar tracts and is connected with the roof nuclei of the cerebellum.

There are also efferent tracts from the superior and inferior colliculi called the *tecto-spinal tracts* (Fig. 69). These probably coördinate eye and body movements with visual and auditory afferent impulses.

Sufficient is known of the physiology and pathology of each of these afferent and efferent tracts to make them all of the utmost interest to the physician in his efforts to interpret nervous diseases.

The striate body and its connections. The striate body developmentally and physiologically consists of two parts, the palæostriatum (ancient striate body) which is in man the globus pallius, and the neostriatum (new striate body) which includes the caudate nucleus and putamen (Fig. 87a, b, and f).

The neostriatum is a small-celled nucleus. It receives afferent fibers from the thalamus, and normally is probably under cortical control. Its efferent connections probably all end in the palæostriatum.

The paleostriatum, or globus pallidus is a large-celled nucleus. Its main afferent connections are from the thalamus chiefly through the intermediation of the neostriatum, but perhaps also directly. It may also be indirectly connected by afferent paths coming from the vestibular nuclei (*Brain,* 1922, page 455). Its *efferent* fibers end in the red nucleus, the nucleus hypothalamicus (of Luys) and the substantia nigra.

The efferent path from the red nucleus is the rubrospinal tract just described. The efferent paths from the nucleus hypothalamicus and substantia nigra are unknown.

The physiology of these nuclei is still very obscure. The corpus striatum in man and higher mammals appears to represent an automatic motor mechanism as contrasted with the voluntary motor mechanism (pyramidal system). Disease of the neostriatum appears to be associated with uncontrollable involuntary choreoathetoid movements, while disease of the palæostriatum is associated with general muscular rigidity and tremor. Perhaps this is due to lack of pallidal control over the nuclei of the subthalamic region—the nucleus ruber, the nucleus hypothalamicus, substantia nigra and perhaps others.

EXAMINATION OF CROSS SECTIONS OF THE ADULT SPINAL CORD

In the preliminary examination of cross sections of the spinal cord and brain stem naked eye sections or drawings of the specimens are to be used, as well as series of microscopic sections stained by iron hematoxylin, and examined under a magnifying lens (× 6) (Figs. 1 to 5).

(1) Throughout the cord the white matter is peripheral, the gray matter is central, and there is a general similarity of pattern in all regions (Figs. 1 to 9, 53 to 55). The cord like the rest of the nervous system is bilaterally symmetrical; it is partially divided into two lateral halves by an anterior median fissure and by a posterior median septum. The anterior median fissure (Figs. 1 to 9) only penetrates approximately one-third of the thickness of the cord; it is well marked. Into it passes a fold of pia mater carrying vessels. At the bottom of this fissure is the white commissure which should be called the white decussation, as it consists of decussating and not commissural fibers. The posterior median septum is formed of neuroglia only. It can sometimes be seen with a low power lens (× 10). It reaches halfway into the cord, where it meets the gray commissure. In the cervical region it may be possible under a low power lens to differentiate the posterior column into the fasciculus gracilis medially and the fasciculis cuneatus laterally (Figs. 1 to 3). There is on the whole a steady increase of white matter as one ascends in the cord. As the white column consists mainly of afferent fibers from the periphery passing directly or by relays to the brain, and of efferent fibers from the brain passing directly or by relays to the periphery, this steady increase of fibers from the lower to the upper end of the cord follows as a matter of course. In the region of the great plexuses, however, there is

a heaping up of short afferent axons, and also of association tracts between neighboring regions of the cord, which somewhat masks this steady increase of fibers from below upward, and partly accounts for the cervical and lumbar enlargements.

The spinal gray matter is central. In section it presents the form of two crescents united by a gray commissure, and it has long been customary to say that each of these crescents represents an anterior horn and a posterior horn. Modern terminology, however, recognizes that what appear as the horns of a crescent in cross section are really gray columns, and we now speak of the anterior and posterior gray columns (columnæ griseæ). Throughout the cord the anterior gray column is the more massive of the two. The anterior gray column does not nearly reach the surface of the cord and its nerve roots must cross the peripheral white matter before emerging. In suitably cut stained sections, examined under a lens, these roots can be seen in three or four groups of fila arranged transversely, passing from the anterior surface of the gray column to the surface of the cord. The difference in size of the anterior and posterior cell columns is especially apparent in the two enlargements or intumescentiæ (Figs. 2 and 4, 6 and 8). The anterior gray columns contain among other things the cell bodies of the lower motor neurons with commencing axons and their dendrites, and the many axon terminals and collaterals which synapse with these dendrites. It is apparent therefore that where the limb muscles are represented by motor neurons there must be a great heaping up of gray matter. This may be demonstrated by contrasting the anterior gray columns in the intumescentiæ (Figs. 2 and 4) with those in the thoracic segments of the cord (Fig. 3), comparing unstained sections with stained sections examined under a hand lens ($\times$ 6 or 10). In suitably stained preparations the cell groups in the anterior gray columns are readily seen. The anterior gray columns in the thoracic segments (Fig. 3) where only spinal and intercostal or abdominal muscles are represented are very small as compared with those of the lumbar and cervical enlargements. In the lumbosacral segments (Figs. 4, 5, 8 and 9) and in the fifth to the eighth cervical and first thoracic segments (Figs. 2 and 6) the anterior gray column expands laterally into a lateral column where nuclei are found representing the more outlying limb muscles. In the thoracic segments where trunk muscles alone call for motor neurons, only the more medial cell groups are required, and hence only the more medial part of the anterior gray column is represented. In the thoracic region, however (Figs. 3 and 7), a special column of planchnic (visceral) efferent

cells is found forming the spinal efferent part of the sympathetic (autonomic) system. These cells form a distinct lateral projection in a plane just in front of the gray commissure called the intermediolateral cell column. This extends continuously from the first thoracic to the first lumbar segments inclusive. A similar group of cells is found in the first and second cervical and in the third and fourth sacral segments, but here the column on section blends with the lateral projection of the anterior cell column. In the thoraco-lumbar region the axons of these cells form the white rami communicantes to the sympathetic ganglia. In the sacral region, their axons go to the pelvic plexuses of the sympathetic, and in the upper cervical region they form splanchnic efferent fibers to the accessory nerve.

The posterior gray column is united to the anterior column by a narrow neck, the cervix columnæ posterioris. It is covered dorsally by a layer of what is called substantia gelatinosa (of Rolando). In the regions of the great plexuses this is crescentic; in other regions, notably in the thoracic segments, it is V-shaped. This is called the caput columnæ posterioris (head of the posterior column). The gelatinous appearance which accounts for the name is seen only in fresh, unhardened sections. In formalin specimens the color of this cap differs little from that of the white matter. It can best be identified in specimens stained by Pal-Weigert's method or by the iron hemotoxylin stain for myelin where its lack of stain makes it prominent (Figs. 1 to 5). The amount of gelatinous substance appears to bear a direct relation to the extent of skin area supplied by the afferent nerves of the segments. Thus it is abundant in the region of the lumbar and sacral plexuses and of the cervical plexus, and in the medulla oblongata and upper cervical segments of the cord (Figs. 56 to 60), where it forms one of the end nuclei of the sensory root of the fifth cranial nerve. The substantia gelatinosa consists of many small nerve cells and a few cells of intermediate size imbedded in much neuroglia. The small cells are of the Golgi type II variety and are probably short association neurons. The larger cells probably form by their axons the secondary afferent tracts for heat, cold, and pain to the thalamus. Just superficial to the caput columnæ posterioris there is a thin white column called the posterolateral fasciculus (Lissauer's column). Dorsomedial to this the posterior nerve roots enter the cord in single bundles (Figs. 2, 4 and 5) which correspond in vertical series with the posterolateral groove. As in the case of the anterior nerve roots, the nerve fila

are seen in only the few sections of a series which happen to cut through them.

Dorsal Nucleus (Nucleus dorsalis.) In the first lumbar segment on the medial side of the neck of the posterior gray column and just behind the gray commissure there is a distinct swelling which marks the lower end of the nucleus dorsalis (O. T. Clarke's column) (Figs. 3 and 7). This is a very prominent collection of large nerve cells, second in size among those in the cord to the motor cells of the anterior gray column. The nucleus dorsalis extends upward to the second thoracic segment, but it is only in the lower six thoracic and first lumbar segments that it produces a distinct bulging of the gray matter. Higher up it is submerged. In specimens stained by myelin stains fibers can be seen entering this nucleus from the deep and lateral part of the dorsal white column (Fig. 54). These fibers end round the cells of the nucleus. In the first lumbar segment there is a very great excess of fibers, most of them longitudinal, which come from the cord lower down. In the cord of a patient who lived three and a half years after extensive destruction of the lumbosacral region of the cord the author found marked degeneration of the dorsal nucleus at a distance of 7.5 cm. above the crush. The axons of the cells of the nucleus dorsalis form the dorsal spinocerebellar tract to be described later. In the lumbar and cervical regions isolated cells forming the cervical and lumbar nuclei of Stilling, have the same connections as those of the nucleus dorsalis.

In addition to the special collections described there are many cells scattered throughout the gray matter which cannot be grouped for descriptive purposes. Some of them form tracts to the brain and others are the cell bodies of association neurons which form the fasciculi proprii and connect different segments of the cord (Fig. 69).

The gray commissure unites the two gray columns of the cord. In the lower lumbar and sacral segments (Figs. 4, 5 and 53) it is two to four times as thick as the white commissure; elsewhere it is very narrow (Figs. 1, 2 and 3.) In its center is the central canal. It consists mainly of neuroglia with fine myelinated and nonmyelinated fibers, crossing in front of and behind the central canal.

The central canal. From end to end of the cord the central canal can be seen in the middle of the gray commissure. Sometimes it is just visible to the naked eye, but it is better seen by aid of a lens. Its cavity is often filled with cells which may obscure it. In the conus medullaris it

approaches the dorsal surface of the cord and may lie under the pia. About the middle of the oblongata (Fig. 11) the central canal opens out to form the floor of the fourth ventricle. It is lined by ependymal cells and is surrounded by neuroglia.

Large branches of the anterior spinal vessels can be seen with a lens passing into the neck of one or other of the anterior gray columns by piercing the commissures from the bottom of the anterior median fissure.

In the second and third cervical segments (Figs. 1 and 56), the gray matter on section is in shape much like the gray columns of the thoracic region, but the size of the whole cord and the large size of the white columns as compared with the gray differentiate these segments. The intermediolateral cell column appears here affording visceral efferent roots to the accessory nerve (Figs. 1 and 56). The presence of this column, coupled with the small size of the somatic motor roots of these segments, which supply trunk muscles only, accounts for the shape and size of the gray columns here (Fig. 1). The gelatinous substance, however, is more abundant. On its surface the posterolateral column is replaced by the spinal tract of the fifth nerve (Figs. 1 and 56).

In the first cervical segment (Fig. 56; note: this figure is probably the upper end of the second cervical segment) the gelatinous substance is abundant. Here it forms part of the sensory nucleus of the fifth nerve, whose spinal tract lies on its surface and replaces the dorsolateral column as in the second and third segments. The neck of the posterior gray column is very thin. Lateral to the thin neck a deep bay is formed which is occupied by longitudinal white fibers with much interlacing gray matter, chiefly neuroglia. The network of fibers gives rise to the appearance called the formatic reticularis of the spinal cord. It is not well named, as there is no real resemblance to the formatio reticularis of the oblongata and the parts higher up. It is mainly associated with the establishment of the lateral position of the lateral cerebrospinal fasciculus.

The many tracts in the cord cannot be identified at present and will be studied later by special methods, but their location should be fixed in mind now by comparing the adult cord with the fœtal cords shown in Figure 69 and Figures 53, 54 and 55. Some of the tracts are differentiated in these by late or early myelination. The sooner one becomes familiar with the names and locations of the tracts the better.

SECTIONS THROUGH THE MEDULLA OBLONGATA

The medulla oblongata (frequently called the bulb) can only be

understood if the naked eye appearance be constantly compared with stained sections examined by a hand lens. With naked eye unstained sections and microscopic sections stained for myelin and with the diagrams before him, the student should be able to understand the meaning of the appearances presented by the sections.

In the discussion the shorter term bulb will often be used for the medulla oblongata.

The central canal and central gray matter. Comparison of the successive sections from below upward, and of Figures 58 to 60, 15 and 16, shows that the central canal of the spinal cord is at first continued upward into the bulb in much the same relative position as it occupied in the cord, but it gradually shifts backward till it finally expands into the fourth ventricle. It is hence common to speak of the "closed" and "open" parts of the bulb. Simultaneously with the opening up of the central canal the central gray matter spreads out on the floor of the fourth ventricle. This redistribution of the central gray matter of the cord is at first difficult to understand. The large motor cells corresponding in series with those in the ventrolateral part of the anterior gray column of the cord are continuous with a column of cells imbedded deeply in the substance of the bulb called the nucleus ambiguus (Figs. 14, 15, 57, 58 and 59; compare also Figs. 12 and 13). This forms the somatic motor nucleus of the ninth, tenth, and eleventh cranial nerves, and is in series with the motor nucleus of the seventh nerve a little higher up.

A column of large cells at first ventrolateral to the central canal is in series with the posteromedial group of motor cells in the first cervical segment. It is the motor nucleus of the twelfth cranial nerve, the motor nerve to the tongue (Figs. 14, 59, 12 and 13). Traced upward it gets to a position lateral to the central canal; and as the canal opens out into the lower end of the median groove in the floor of the ventricle the nucleus comes to lie in the floor of the ventricle immediately lateral to this groove. Here it corresponds in position to the trigonum hypoglossi (Fig. 11). From its ventral aspect emerge the fila of the hypoglossal nerve to assume a ventral course medial to the inferior olive. This column is in series with the nuclei of the sixth, fourth, and third nerves higher up (Figs. 12 and 13).

In the floor of the fourth ventricle at its lower end, lateral to the hypoglossal nucleus in the region of the trigonum vagi (Fig. 11), is a column of cells smaller in size than the cells of the nucleus hypoglossi. This column, rounded on section, forms the dorsal nucleus of the vagus

and glossopharyngeal. It is mixed in character and contains the cell bodies of visceral efferent neurons to the unstriped muscle supplied by the vagus and glossopharyngeal nerves, and smaller cells forming the end nucleus of visceral afferent neurons in the same nerves. Lateral to this column is a well marked, rounded bundle of longitudinal fibers called the fasciculus solitarius. This is the spinal tract of the glossopharyngeal nerve and nervus intermedius, and the cells in its neighborhood form the end nucleus of the taste fibers in these two nerves. The visceral efferent nucleus of the vagus and glossopharyngeal corresponds in series with the intermediolateral cell column in the thoracic cord and second cervical segment. The visceral efferent nuclei of the seventh and third nerves are not, apparently at least, in series with the lower visceral efferent nuclei. The issuing fila from these nuclei will be considered later.

Cerebrospinal tract. The great cerebral voluntary motor tract, the tract of the upper motor neurons, commonly called the pyramidal tract, appears at the lower end of the oblongata as the *pyramidal decussation* (Figs. 10 and 58). This occupies the place of the anterior median fissure of the cord. Above this level we find two concentrated tracts, one on each side of the anterior median sulcus (Figs. 59 and 60). These are the *pyramids* or *cerebrospinal tracts* before the decussation. At the decussation the medial three-fourths, more or less, of each pyramid decussates with its fellow to lie deeply in the cord lateral and anterior to the posterior gray column. Here it is called the *lateral cerebrospinal fasciculus* (O. T. crossed pyramidal tract). Compare Figures 59 to 55 from a fœtus at term with each other where the pyramids and cerebrospinal tracts are well differentiated by their late myelination. In fœtal specimens it is also seen that in the cervical and thoracic regions of the cord the lateral cerebrospinal tract is separated from the surface by a tract called the posterior spinocerebellar tract, distinct because of its early myelination. In the lumbar region, on the other hand, the lateral cerebrospinal tract lies on the surface because the posterior spinocerebellar tract only commences in the lower thoracic segments. This is also rendered evident by degeneration of the tract (see Fig. 79, j, k and l). The whole pyramid does not usually cross at the pyramidal decussation. The lateral fourth, more or less, runs downward on the same side of the cord close to the anterior median fissure, forming the *anterior cerebrospinal tract* (O. T. direct pyramidal tract) (Figs. 55, 56 and 79, k, l, m, n). This is, however, only a delayed decussation, the fibers crossing one by one and the tract usually ending in the

upper thoracic region. Sometimes the whole pyramid crosses at once in the medulla oblongata. More often the anterior cerebrospinal tract can be traced as low as the sacral segments (Fig. 79, l, m and n).

The ending of the funiculus gracilis (O. T. column of Goll) in the *nucleus gracilis* and of the funiculus cuneatus (column of Burdach) in the *nucleus cuneatus* may next be noted. If one examines successive sections from below upward (Figs. 57 to 59) the funiculus gracilis is seen to be gradually invaded on its deep surface by gray matter, and so also is the funiculus cuneatus, though the invasion begins a little higher up. The swellings called the clava and tuberculum cuneatum are thus produced (Fig. 11). From the ventral surface of these nuclei curved fibers emerge called the *internal arcuate fibers* (Fig. 59). They start in the cells of the gracile and cuneate nuclei, and decussate with each other. Those of each side form the opposite *medial lemniscus* (Figs. 59 and 60) which soon becomes a structure prominent even to the naked eye in unstained sections and still more in stained sections, lying on each side of the median raphe of the oblongata between the inferior olivary nuclei (Fig. 60). This is the relay for the long fibers of the posterior columns of the cord to the opposite side of the thalamus. It carries muscle sense and tuning fork sense to the thalamus, but tactile discrimination separates here and travels by fibers more laterally placed in the formatio reticularis.

Just ventral (ventrolateral or directly lateral at some levels) to the nucleus cuneatus is the *gelatinous substance* (Figs. 58 and 59) capped on its surface by the crescentic *spinal tract of the fifth or trigeminal nerve.* As has been seen, this extends downward into the upper segments of the spinal cord. Some of the curved fibers which cross through the oblongata from the medial side of the gelatinous substance become longitudinal in the opposite formatio reticularis; they form the *trigeminothalamic tract* (Fig. 15). These are the second sensory neurons for pain, heat, and cold for the sensory root of the trigeminal nerve. Their cell bodies are in the gelatinous substance and they end in the opposite thalamus.

Lateral to the spinal tract of the fifth nerve a well-marked longitudinal tract is gradually formed increasing in size as it approaches the upper end of the medulla oblongata. This is the *restiform body.* At first it is formed by the shifting backward of the *dorsal spinocerebellar tract* (Figs. 57, 58 and 59), which is at first concentrated into a triangular bundle just ventral to the spinal tract of the fifth nerve (Figs. 57 and 58). The restiform body is joined on its dorsal surface by posterior external arched

fibers from the accessory cuneate nucleus (Fig. 59). Later it receives a great accession from the opposite inferior olive (Figs. 60 and 14). As it is traced upward, it goes to the vermis of the cerebellum (see Fig. 60).

In sections through the middle of the medulla oblongata, the *inferior olive* is a very prominent structure (Figs. 59 and 60; compare Figs. 10, 14 and 15). It is a much folded sheet of gray matter, open medially. Two small flat sheets of similar gray matter appear in sections as bands situated respectively medially and dorsally to the olive. They are the medial and dorsal accessory olives (Fig. 14). For the mode of folding of the olive see Cunningham, Figure 460. Dorsal to the olive and between it and the dorsal accessory olive are seen many longitudinal fibers cut across (Figs. 78 and 15). They consist partly at least of spino-olivary fibers derived from the endogenous fibers of the cord and coming from cell bodies in the anterior gray columns, and they end in synapses with the olivary cells. From the hilum of each olive (Figs. 60 and 14) the axons of the olivary cells run toward the raphe, pass through the medial lemniscus, intercross in the raphe with their fellows, and run ventral to the spinal tract of the fifth nerve and through this to join the restiform body. They pass to the cerebellar hemisphere and probably by this means each cerebellar hemisphere is brought into relation with the body muscles by way of the spino-olivary and olivocerebellar neurons. It will be observed that this is a crossed afferent cerebellar connection. It is the only crossed peripheral-afferent cerebellar connection known to the author, and no crossed cerebellar efferent tract corresponds with it.

Other olivary connections are less definite, and though the olives like the cerebellar hemispheres are best developed in man, and though each olive degenerates with the opposite cerebellar hemisphere should that be diseased, their special function is unknown.

The anterior or ventral spinocerebellar tract (Figs. 69 and 53 to 60) is easily distinguished with a lens in stained sections as a triangular tract somewhat loosely scattered ventral to the dorsal spinocerebellar tract in the bulb. It soon passes deeply into the pons, where it can only be distinguished when degenerated and stained by the Marchi method. It may be found again on the surface of the brachium conjunctivum (Fig. 64); by this circuitous route it reaches the vermis. It contributes fibers to the nuclei of Deiters and Bechterew. Mediodorsal to the ventral spinocerebellar tract are the cells of the nucleus lateralis (Fig. 59). The function of this nucleus is unknown.

OTHER LONGITUDINAL TRACTS IN THE MEDULLA OBLONGATA

The medial longitudinal fasciculus is a very prominent bundle of early myelination and great physiological and clinical importance. It lies in the floor of the fourth ventricle, close to the median groove (Figs. 59 to 68). Below the pyramidal decussation it is continuous with the anterior fasiciculus proprius of the cord, and in the mesencephalon it is found near the floor of the aqueductus cerebri. It is easily distinguished as a dark concentrated band of rather large fibers in the situation indicated; and in fœtal specimens it is made prominent by its early myelination (Figs. 59 to 68). Its origin and function will be studied in connection with Deiters' nucleus.

The *tectospinal tract* may be recognized in stained adult specimens as a tract of smaller and less densely packed fibers between the medial longitudinal fasiciculus and the medial lemniscus. In fœtal specimens it myelinates late (Figs. 59, 60, and 15). It runs from the corpora quadrigemina to the lower motor neurons, and in the cord is found in the anterolateral column (Fig. 69). It probably coördinates muscular action with visual and auditory impressions.

The rubrospinal, vestibulospinal, spinothalamic, and trigeminothalamic, and spinotectal tracts cannot be differentiated except by special experimental methods. Their location in man is inferred as corresponding in a general way with the position as determined by experiment in monkeys. This is confirmed by clinical symptoms and pathological findings in man. They all occupy an area medial to the gelatinous substance and dorsal to the ventral spinocerebellar tract.

The formatio reticularis. In stained sections one can easily see fibers dorsal to the olives crossing each other in nearly all directions, producing a network which has received the general name of formatio reticularis. Medial to the emerging roots of the twelfth nerve this network is almost devoid of nerve cells and is called the white reticulation (reticularis alba); lateral to the twelfth nerve roots there are many nerve cells scattered and in groups, and the formation is consequently spoken of as the gray reticulation (reticularis grisea). (See Figs. 59 and 60.) The functions of the cells scattered through the reticular formation are not definitely known. Many endogenous fibers of the cord, as well as endogenous fibers from higher up in the brain stem end here. Physiologically the medulla oblongata is rich in centers. The respiratory, gastric, and cardiac centers may possibly be associated with the vagus and accessory nuclei; while the

diabetic, vasoconstrictor, pupil-dilating, and oculo-rotary centers may possibly be associated with the more scattered cells of the reticular formation.

The *median raphe* of the bulb consists very largely of decussating fibers. Of these we have described internal arcuate fibers from the gracile and cuneate nuclei to the opposite medial lemniscus; fibers from the gelatinous substance to the opposite trigeminothalamic tract, fibers from the inferior olives to the opposite restiform bodies, and fibers from the glossopharyngeal sensory nucleus to somewhere unknown in the opposite formation reticularis.

In suitable sections fibers may be seen passing from the pyramid dorsalward in the raphe, and crossing immediately dorsal to the medial longitudinal bundle to the opposite twelfth nucleus. This is probably the general direction taken by pyramidal fibers crossing to motor nuclei of other cranial nerves.

The anterior external arcuate fibers also emerge from the ventral surface of the raphe. They pass round the ventral surface of the pyramid and olive to the restiform body. Associated with them is a nucleus situated on the superficial surface of the pyramid known as the *arcuate nucleus* (Figs. 59 and 15). They are aberrant fibers from the basilar portion of the pons and belong to the cortico-pontine system.

Cranial nerves and their nuclei in the medulla oblongata. (Figs. 10, 11, 12, 14, 15, 59, and 60). The fila of the *hypoglossal nerve* or twelfth cranial nerve are very prominent in the stained sections of the bulb. They emerge from a nucleus close to the central canal below (Figs. 14 and 59), and close to the median sulcus of the floor of the fourth ventricle higher up. Their course is ventral and slightly lateral. They pass medial to the olive and lateral to the pyramid and partly through both of these structures. They emerge as a dozen bundles, more or less, along the medial border of the olive. (Figs. 59 and 10.)

The glossopharyngeal, vagus, and accessory nerves (ninth, tenth, and eleventh cranial nerves) (Figs. 10, 14, 15, and 57, 58 and 59) form a series of bundles which pass ventrolaterally through the spinal tract of the fifth nerve or between it and the restiform body. The *nucleus ambiguus* (Figs. 59, 14, and 15) is the motor nucleus of these nerves to pharyngeal and laryngeal muscles. Even under a hand lens fibers can be seen in stained sections streaming dorsalward from this nucleus. They afterward bend laterally and ventralward and become grouped together in the emerging fila of the motor roots (Fig. 14). The root of the accessory nerve extends

down for some distance in the spinal cord. Its fibers emerge laterally (Figs. 56 and 57). The visceral efferent and visceral afferent nucleus of the vagus and glossopharyngeal will be seen afterward lying lateral to the hypoglossal nucleus. In connection with the glossopharyngeal nerve there is an interesting rounded bundle of fibers which is prominent even in naked eye unstained sections of the bulb. It is the *fasciculus solitarius* (Figs. 14, 15, and 59). It occupies a dorsal position, at first medial to the internal arcuate fibers from the gracile nucleus, and higher up medial to the restiform body. Nerve fila can be seen running into and encircling it and its neighborhood is occupied by its nucleus. This is the spinal tract of the glossopharyngeal nerve; its nucleus is the gustatory nucleus. The taste fibers of the chorda tympani and vagus nerves join this tract.

The spinal tract of the vestibular nerve (Figs. 14, 15, and 60) is seen in sections of the bulb at the level of the lateral ventricular aperture. It extends downward for a short distance. It is recognized as scattered bundles of fine fibers dorsal to the fasciculus solitarius and medial to the restiform body.

The lateral apertures of the fourth ventricle, the chorioid plexuses, tæniæ and ependyma of the roof. At the correct level, if the cerebellum is retained in the sections, one can see the lateral aperture of the fourth ventricle, the little tuft of chorioid plexus which emerges from it, and the thinned-off roof of the medulla oblongata, which forms the tænia ventriculi quarti. These are all well shown under suitable powers and can partly be made out in unstained, naked-eye sections.

A section through the upper part of the medulla oblongata, running slightly upward and backward, a little above the lateral aperture of the ventricle, is drawn in Figure 60. Such a section should be examined if possible with a hand lens and later with higher powers.

The pyramids, medial lemniscus, olive, anterior spinocerebellar tract, spinal tract of the fifth nerve, with the gelatinous substance, and the emerging fila of the twelth nerve should be recognized, but require no special description. In this section (Fig. 60) the olivary fibers of the restiform body are well shown. The restiform body itself is seen in its passage backward round the outside of the corpus dentatum cerebelli to the vermis of the cerebellum. The special feature of this section is the vestibular nerve and its connections. In the figure the vestibular nerve passes backward between the spinal tract of the fifth nerve and restiform body to several groups of nerve cells in the lateral part of the floor of the fourth ventricle in the neighborhood of the acoustic tubercle. Scat-

tered longitudinal fibers also appear which have been seen in sections lower down (Fig. 59) and which form the spinal tract of the vestibular nerve. The large-celled lateral nucleus called Deiters' nucleus is of special interest. The vestibular nerve partly ends here. In suitable sections fibers can be seen passing from the dorsolateral side of Deiters' nucleus and running dorsally, medial to the dentate nucleus, to the nucleus fastigii and perhaps the nucleus globosus in the roof of the fourth ventricle. From the medial side of Deiters' nucleus, fibers pass:

(*a*) Medioventrally, to a position medial to the gelatinous substance. These form the vestibulospinal tract which later occupies a ventral position in the spinal cord (Fig. 69).

(*b*) Medially, to the homolateral sixth nucleus and to both medial longitudinal bundles of which they form the main constituent. The fibers from Deiters' nucleus which pass by way of the medial longitudinal bundle to the head-turning motor nuclei (the nuclei of the accessory and the first four cervical nerves) are mainly homolateral; those to the medial longitudinal fasciculus which go upward to the nuclei of the third cranial nerve are many and are mostly crossed. A few go upward by the homolateral medial longitudinal bundle to the nucleus of the fourth cranial nerve. All these connections are of the utmost clincial importance. After contributing fibers to the nuclei for the eye muscles the medial longitudinal bundles end in the interpeduncular nucleus and from this relays pass to the globus pallidus of the corpus striatum.

SECTIONS THROUGH THE PONS

The best sections through the pons are (*a*) at the lower end through the lower border of the pons and the striæ medullares; (*b*) through the middle of the pons so as to include the roots of the fifth nerve (this section should by preference run parallel with the upper border of the brachium pontis); and (*c*) through the upper end of the pons, including the superior medullary velum just below the inferior colliculi so as to show the emerging fourth nerve.

Even naked-eye sections of the pons show that it is readily divided into a *basilar portion* ventrally and a *tegmental portion* dorsally. The former is the continuation downward of the basis pedunculi of the mesencephalon, and the latter is the continuation of the tegmentum mesencephali.

The *pars basilaris pontis* is very similar throughout (Figs. 16, 17 and 62 to 65). At the lower end it is divided from the *pars tegmentalis pontis*

by the *medial lemniscus* and *corpus trapezoideum,* and at the upper end by the medial and lateral lemnisci.

In Figures 63 to 66, showing sections through the pons of a fœtus at term the pyramidal fibers are distinguished by the stage of myelination. They myelinate earlier than fronto-pontine and temporo-pontine fibers. The transverse fibers myelinate still later. The basilar part of the pons consists of longitudinal fibers, cells and transverse fibers. The transverse fibers commence as the axons of the pontine cells and soon acquire their myelin sheaths. Immediately taking a transverse direction they mainly cross the median line to form the opposite brachium pontis. The longitudinal fibers are the direct continuation downward of the basis mesencephali. The middle three-fifths of the basis mesencephali passes through the pons to form the pyramidal tract in the medulla oblongata, losing only a few fibers to motor nuclei of cranial nerves in the pons. The medial and lateral fifths of the basis mesencephali end by arborizing round the cells of the basilar part of the pons. The cells in this are called the nuclei pontis, and their axons form the transverse fibers of the pons already described.

At the upper end of the pons the *basilar fibers of the basis pedunculi* enter as a series of longitudinal bundles divided by the transverse fibers of the pons (Figs. 17, 64 and 65).

The medial and lateral fifths of the basis pedunculi (Fig. 67) are formed of *fronto-pontine* and *temporo-pontine* fibers respectively. As their names imply they end in the pons by synapsing with the cells of its basilar part. The axons of these cells cross the median line and converge to form the opposite *brachium pontis* (Figs. 16 and 64), the fibers of which end in the cortex of the cerebellar hemisphere by synapsing with the cells of Purkinje (Fig. 21). Thus there is formed a path with a relay in the pons between the cortex of one hemisphere of the cerebrum and the cortex of the opposite hemisphere of the cerebellum.

The middle three-fifths of the basis pedunculi consists of *pyramidal fibers* carrying voluntary motor impulses to the cells of the lower motor neurons. In their passage through the pons the pyramidal bundles give off collaterals to the nuclei pontis and upper motor neuron fibers to the motor nuclei of both fifth nerves, but mainly to that of the opposite side. A little lower they give fibers to the opposite sixth nucleus, and to both seventh nuclei; the homolateral fibers to the seventh nucleus, however, supply only the upper part of the face. For the lower face muscles all the pyramidal fibers are crossed. Hence it follows that in an upper motor

neuron facial paralysis the muscles of the upper part of the face (orbicularis oculi, forehead muscles) are only weak because of the bi-lateral pyramidal supply. The lower part of the face is, however, completely paralyzed, because it has only a crossed pyramidal supply. On the other hand, a lower motor nueron paralysis of the face muscles involves the whole side of the face. The same distinction between upper and lower motor neuron paralysis applies to the larynx, palate, pharynx, and tongue, all of which have a homolateral as well as a crossed pyramidal supply.

As the pyramidal fibers approach the lower end of the pons, they are gathered into a single compact bundle on each side preparatory to entering the medulla oblongata (compare Figs. 63, 62, 61 and 60, pyramid).

Tegmental part of the pons (*pars tegmentalis pontis*). As the *medial lemniscus* enters the lower end of the pons it changes from the ribbon-like band seen in the medulla oblongata to a somewhat prismatic form on each side of the middle line (compare Figs. 60, 61, 62, 63, and 64).

Farther up it becomes flattened out and shows in transverse sections as a nearly horizontal band. At the upper end of the pons it is joined by the spinothalamic and trigeminothalamic tracts and there appears laterally to it a band of finer fibers called the *lateral lemniscus*. This is the relay tract for the nerve of hearing (cochlear nerve) to the inferior colliculus and medial geniculate bodies (Figs. 64 and 17).

The cochlear nerve and trapezoid body (*corpus trapezoideum*). (Fig. 61, compare with Fig. 102.) Lateral to and a little lower than the vestibular nerve the cochlear nerve ends in two nuclei; one of these is ventrolateral to the corpus restiforme at the upper limit of the medulla oblongata, the other is dorsolateral to it. From the inner side of the *ventral nucleus* a new relay of fibers passes obliquely upward and inward to the lower end of the pons. This forms a very distinct band of deep transverse fibers of early myelination called the corpus trapezoideum. It crosses through the medial lemniscus, decussates with its fellow of the opposite side and forms the *lateral lemniscus* which takes an upward direction ventral to the commencing spinal tract of the fifth nerve (Figs. 62 and 63). Later this is found at the upper end of the pons lateral to the medial lemniscus (Figs. 63 to 68). The *corpus trapezoideum* is a prominent structure in naked-eye and microscopic sections of the lower end of the pons (Fig. 63).

In sections through a suitable level the striæ medullares show passing from the dorsal nucleus as a well-marked band (or bands) of fibers crossing inward on the floor of the fourth ventricle (compare Figs. 11 and 61

with 102) till it reaches the middle line where it dips into the median groove and decussates with its fellow of the opposite side (see Fig. 11). Its further course is difficult to trace, but it joins the corpus trapezoideum opposite to the side from which it springs (Fig. 102). Clinical evidence, however, shows that there are uncrossed fibers passing from the cochlear nuclei to the lateral lemniscus of the same side, for one-sided destruction of the secondary and tertiary tracts for hearing does not produce deafness of the opposite ears. (Dejerine states that the crossing is complete.)

The *superior olive* is a rather large irregular nucleus developed in the corpus trapezoideum (Figs. 62 and 63) ventromedial to the nucleus of the seventh nerve and ventromedial to the spinal tract of the fifth nerve. It is surrounded by many longitudinal fibers seen in cross section. The significance of these is unknown. Many of the fibers of the corpus trapezoideum are connected with it either directly or by collaterals (compare Fig. 102), and from its dorsal surface fibers stream in a dorsomedial direction toward the nucleus of the sixth nerve (Fig. 63). These form the *peduncle of the superior olive* and seem to form a short path by which the head and eyes may be turned reflexly toward the side on which a sound is heard.

The medial longitudinal fasciculus is a well-marked concentrated band close to the median line in the floor of the fourth ventricle. Ventrolateral to this fasciculus just above the striæ medullares at the lower end of the pons is a short column of large cells which form the *nucleus of the sixth or abducent nerve* (Fig. 63). The fila of the sixth nerve issue from the medial side of the nucleus and immediately turn ventrally in numerous bundles which run through the medial lemniscus and pyramid to emerge at the lower border of the pons near the middle line. The nucleus of the sixth nerve is connected with the opposite pyramidal tract, with Deiters' nucleus, and with the superior olive, and contains cells which send fibers to the homolateral medial longitudinal bundle. These ascend in the medial longitudinal bundle to synapse with the cells of the nucleus of the third or oculomotor nerve in the mesencephalon and thus provide for coördinated action of the opposite internal rectus with the homolateral external rectus oculi in conjugate deviation of the eyes. This is an indirect action, as no fibers from the medial longitudinal bundle enter the third nerve.

The seventh nucleus. (Figs. 12, 13, 24, 62 and 63). Medial to the spinal tract of the fifth nerve and dorsolateral to the superior olive is a space which appears clear under low powers in stained sections, but which

under higher powers shows the large cells that form the motor nucleus of the seventh or facial nerve. Scattered fila of origin spring from its dorsal surface, run dorsomedially to the floor of the ventricle, and then cross medially over the sixth nucleus to a position dorsolateral to the medial longitudinal bundle (Figs. 24 and 62). Here they become concentrated in a short longitudinal bundle. From this bundle the emerging roots run lateralward and then ventralward between their own nucleus and the spinal tract of the trigeminal nerve (Fig. 62) to emerge at the lower border of the pons just medial to the spinal tract of the fifth nerve. The writer has not been able to trace the nervus intermedius or sensory root of the seventh nerve. Its function is gustatory for the anterior two-thirds of the tongue, and it joins the fasciculus solitarius.

The fifth nerve and its nuclei (Figs. 10, 12, 13, 16, 62 and 63). It is impossible except in a complete series of sections to see satisfactorily the fifth nerve and its nuclei. The nerve on entering the pons passes dorsalward and slightly caudalward toward the floor of the fourth ventricle and the lower end of the pons. Figure 63 shows it well and with the help of this figure the fifth nerve nuclei should be easily recognized in stained sections. Under a low power the roots of the nerve are seen to pass dorsally, lateral to the seventh nucleus, to end in two distinct nuclei, one dorsomedial and one lateral. Under high powers in suitably stained sections, these prove to be the motor and the lateral sensory nuclei. The motor nucleus is a group of large multipolar cells dorsal to the seventh nucleus. The lateral sensory nucleus is ventral to the brachium conjunctivum. It reminds one in its general structure of the nucleus gracilis; with this the writer believes it homologous, that is, he believes it to be the end nucleus of the fifth nerve for conscious muscle sense for the muscles of mastication and very probably also for the muscles of the face and tongue. The lateral sensory nucleus is connected with the vermis cerebelli, and is relayed probably by the opposite medial lemniscus to the thalamus and thence to the cerebral cortex. *The spinal tract of the fifth nerve* is formed by descending fibers from the sensory root. It extends from the level of the lower pons to the second cervical segment of the spinal cord. It consists of fine myelinated fibers and very many non-myelinated fibers. The latter convey heat, cold, and pain; the former probably convey light touch. Those fibers that travel farthest down the spinal cord represent a zone of skin on the head and face extending from the vertex, down in front of the ear to the chin (see Fig. 96a). Successive zones approaching the nose represent successively higher levels of

nerve endings. Section of the spinal tract of the fifth nerve at the level of the inferior olive causes complete anæsthesia of the cornea and loss of pain, heat, and cold in the face behind this. It is to be noted that the cornea is only sensitive to pain; tactile stimuli of all kinds are interpreted as pain. This tract is a prominent crescentic band which forms an important landmark in all sections of the lower pons or oblongata. Medial to it is the gelatinous substance in which its fibers end. The spinal tract and gelatinous substance are seen commencing in Figure 63 (see also Figs. 62 to 56). In Figure 62 the emerging seventh nerve is seen to lie medial to it; in Figure 60 the vestibular nerve runs between it and the restiform body. This position is distinctive of the vestibular nerve. A little lower the glossopharyngeal-vagus group of nerve roots runs lateral to or through it (Figs. 14 and 15). Medial to the gelatinous substance in the oblongata are the rubrospinal, vestibulospinal, and spinothalamic tracts.

The gelatinous substance which is the trigeminal nucleus for heat, cold, and pain is relayed by transverse fibers to a position in the opposite formatio reticularis about midway between the opposite spinal tract of the fifth and the tectospinal tract (see Fig. 15). This is called the *trigeminothalamic* tract and is important clinically in certain lesions of the medulla oblongata, such as softening due to thrombosis of the posterior-inferior cerebellar artery. Like the spinothalamic tracts, it joins the medial lemniscus at the upper end of the pons and ends in the ventrolateral thalamic nucleus, whence it is relayed to the *dorsolateral thalamic nucleus,* and then to the essential organ of the thalamus and cerebral cortex. The sensory nuclei of the fifth nerve are connected by association fibers with the seventh and twelfth motor nuclei for face and tongue reflexes and probably also with the motor nuclei of the brachial plexus. (It seems possible that this may explain the septomarginal bundle in the cervical cord, Figure 56.) The motor nucleus of the trigeminal nerve receives fibers from both pyramidal tracts, but chiefly from that of the opposite side.

The mesencephalic root of the fifth nerve. Running dorsally from the interval between the motor and lateral sensory nuclei of the fifth nerve there are fibers which pass medially to the brachium conjunctivum (Fig. 63) and form a small tract along the side of the upper part of the fourth ventricle and the aqueductus cerebri (Figs. 64, 65 and 66). This tract is associated with a group of cells on its medial surface; its function is unknown.

The locus coeruleus is a dark spot in the lateral recess of the ventricle just above the nuclei of the fifth nerve. On section it is seen that the dark color is due to a considerable group of rather large pigmented cells. They probably have no connection with the fifth nerve and their significance is entirely unknown.

Cerebellar nuclei and brachia conjunctiva. Hitherto only the afferent tracts of the cerebellum have been discussed. It has been shown that the restiform bodies connect the whole skeletal muscular system, except that of the face and eyes, with the cortex of the vermis, that the inferior olive probably connects the skeletal muscles with the opposite cerebellar hemisphere, and that the brachium pontis connects the cerebral cortex of the opposite cerebellar hemisphere. All these are afferent paths.

All *efferent impulses* from the cerebellar cortex leave by way of the axons of Purkinje cells (Figs. 20 and 21). These end in the cerebellar nuclei; the fibers from the hemispheres go to the nucleus dentatus; the fibers from the vermis go to the *nucleus globosus* and *nucleus fastigii,* and perhaps to the *nucleus emboliformis* and *nucleus dentatus.* In order to see the cerebellar nuclei one must study sections passing through the medulla oblongata in which the cerebellum has been kept in place. The *dentate nucleus* (Figs. 19 and 60) is the largest. It is a much folded sheet of gray matter, its open hilum pointing upward and medialward. It develops proportionately with the cerebellar hemispheres and is large in the higher apes and largest in man. In stained sections it shows many multipolar cells, the axons of which form the main bulk of the brachium conjunctivum; the commencing brachium conjunctivum may be seen on its medial side (compare Fig. 60 with Fig. 19). The *nucleus emboliformis* which lies medial to it may be regarded as an isolated portion of the dentate nucleus. The *nucleus fastigii* or *nucleus tecti* is found in the white stalk of the vermis just above the ending of the inferior vermis (Fig. 60). It receives fibers from the vermis and flocculus and sends its efferent axons to the nucleus of Deiters. By many neurologists it is thought to send fibers alongside the nucleus of Deiters to the spinal tract of the vestibular nerve, and by the vestibulospinal tract to the spinal cord. There is a well-marked decussation between the two fastigal nuclei and probably most of their descending fibers are crossed. The fibers to Deiters' nucleus are seen in Figure 60. The afferent and efferent tracts of the nucleus globosus, which lies lateral to the nucleus fastigii, have probably the same connections as those of the latter nucleus.

In sections through the *middle of the pons* (compare Fig. 63 with

Fig. 62) the *brachium conjunctivum* is a very definite band of myelinated fibers which forms the lateral boundary of the fourth ventricle. The two brachia are joined by the *superior medullary velum.* The longitudinal myelinated fibers seen in transverse section in the velum are in part at least fibers from the *anterior spinocerebellar tract* which reaches the vermis by this circuitous route (Figs. 62 and 63). Medial to the brachium conjunctivum is the *mesencephalic root of the fifth nerve* (Fig. 63); lateral to it are fibers which arch round it from the *anterior spinocerebellar tract* and from part of the restiform body, and which go to the vermis (Fig. 60).

In the sections illustrated in Figures 64 and 17 through the *upper end of the pons* above the brachium pontis, the transverse fibers of the basilar part of the pons are seen descending along the side to join the brachium pontis lower down. The longitudinal bundles of the pars basilaris need no special mention. In fœtal sections such as that illustrated in Figure 64, only the pyramidal part of the longitudinal bundles has become myelinated; the fronto-pontine and temporo-pontine bundles myelinate later. Figure 17 is from an adult and here the longitudinal fibers represent fronto-pontine and temporo-pontine fibers which end in the nuclei pontis as well as pyramidal fibers which pass for the most part straight through the pons and which carry voluntary motor impulses to the lower motor neurons.

The medial lemniscus, now a very definite flattened sheet of fibers, is here joined by the spino-thalamic and trigemino-thalamic tracts; they do not intermingle. Thus each medial lemniscus at this level conveys all forms of ordinary sensation from the opposite side of the body, including the head, to the thalamus. Several small rounded bundles of fibers are found imbedded in the ventral surface of the medial lemniscus, near its medial border, at the upper end of the pons (Fig. 64). These are the *aberrant pyramidal fibers* of Dejerine. They are destined for the supply of the motor nuclei of the fifth, seventh, eleventh, and twelfth cranial nerves. They are both homolateral and crossed, chiefly the latter; the sixth nucleus and the lower part of the seventh nucleus receive only crossed fibers. The crossing takes place in small bundles at the level of the nucleus supplied. In the mesencephalon the aberrant pyramidal fibers will be found as a few rounded bundles, lateral to the substantia nigra.

The lateral lemniscus is the upward continuation of the corpus trapezoideum and is therefore the secondary tract for hearing, mainly from the opposite ear. It lies laterally to the medial lemniscus and is seen passing backward over the surface of the brachium conjunctivum on its

way to the inferior colliculus and medial geniculate body. Its fibers are of finer caliber than those of the medial lemniscus. Embedded among them is a nucleus called the *nucleus of the lateral lemniscus;* the function of this nucleus is not known. In man and apes it is continuous with the superior olive and has probably a similar use.

The medial longitudinal fasciculus will be readily recognized but needs no special mention.

The brachium conjunctivum is here submerged and forms the lateral boundary of the pars tegmentalis pontis. On its lateral surface is the lateral lemniscus; more dorsally, the anterior spinocerebellar tract, on its way to the vermis, passes round the surface of the brachium conjunctivum and enters the superior medullary velum. Medial to the brachium conjunctivum is the formatio reticularis and more dorsally the mesencephalic root of the fifth nerve.

The rubrospinal tract and tectospinal tract are among the longitudinal fibers of the formatio reticularis. The rubrospinal tract is dorsal to the medial margin of the medial lemniscus (compare Fig. 83). The relations and significance of the other fibers of the formatio reticularis are not sufficiently well known to be discussed here.

Nuclei of the fourth nerves and the decussation of their fibers. A little higher, under cover of the inferior colliculi (Figs. 66, 64, 22 and 18), is found the *nucleus of the fourth nerve* embedded in the dorsal surface of the medial longitudinal fasciculus. The fila of the nerve pass lateralward from this nucleus, then dorsalward to form rounded bundles which lie lateral to the aqueductus cerebri, descend to the level of the superior medullary velum (Figs. 64 and 18) and decussate in the velum (Fig. 18) before each emerges from its dorsal surface. This is the only nerve that makes a complete decussation. A partial decussation of the third nerve is the only other instance of decussation of the lower motor neurons.

SECTION THROUGH THE INFERIOR COLLICULI

In most series of sections through the brain stem, the section which passes through the inferior colliculi passes also through the upper part of the pons (see Fig. 65 and compare it with Fig. 22).

The *pars basilaris pontis* needs no special description at this level. The *medial lemniscus* is shifting lateralward. The *lateral lemniscus* is shifting backward; many of its fibers end by sending terminals or collaterals which appear to embrace the gray matter of the inferior colliculus in which they end. The function of the inferior colliculus is not sensory.

It appears to be a reflex center for the coördination of eye and body movements with auditory impressions. This it does by way of the tectospinal tract, which will be described in connection with the superior colliculi. The remaining fibers of the lateral lemniscus pass upward to the medial geniculate body.

The fibers of the *brachia conjunctiva* are here seen to assume a medialward direction and in this section (Fig. 65) their ventral fibers are commencing to intercross.

The next section (Figs. 66 and 22) through the upper part of the inferior colliculi shows the *decussation of the brachia conjunctiva.* This decussation is complete.

If the section be made, as in Fig. 22, at a right angle to the long axis of the brain stem and passes entirely through the mesencephalon, the basis pedunculi will be found separated from the tegmentum mesencephali by a sheet of gray matter. This gray matter is dark in naked-eye sections, since the cells of this *substantia nigra* contain pigment. Many fibers from the lentiform nucleus and basis pedunculi enter it. The function of the substantia nigra is not yet known, but atrophy or chromatolysis of its cells has been found in cases of paralysis agitans (Revue Neurolog., Aug., 1922). Its cells are efferent in type and it probably forms an efferent relay station for the subcortical motor mechanism. The substantia nigra can be traced upward into the subthalamic region.

The *basis pedunculi* consists almost entirely of longitudinal fibers derived from the cortex cerebri. The middle three-fifths are pyramidal fibers, coming from the Betz cells of the anterior central convolution and destined for distribution to the opposite lower motor neurons (compare Fig. 79, c). The pyramidal fibers are much mixed by the time they reach the basis pedunculi, but some grouping remains; the face fibers are mostly medial, the arm fibers intermediate, and the leg fibers lateral.

The medial fifth of the basis pedunculi consists of fibers which come from the frontal lobe, especially the orbital convolutions, and end in the nuclei pontis. A few *aberrant pyramidal fibers* destined for cranial nerve nuclei frequently spread along the ventral border of this part of the basis mesencephali (Fig. 79, c). The lateral fifth of the basis pedunculi comes from the middle or inferior temporal gyrus and also ends in the nuclei pontis. The pyramidal fibers give collaterals to this nucleus. The axons of the cells of the nuclei pontis form the brachium pontis and end by synapsing with the Purkinje cells of the cortex of the cerebellar hemisphere. This connection is almost entirely crossed.

Just above the mesencephalon pyramidal fibers are given off to the opposite oculomotor nucleus. This supply is entirely crossed (Van Gehuchten). Dorsal to the lateral portion of the basis pedunculi are small rounded bundles of pyramidal fibers which lie lateral to the substantia nigra and are destined for distribution to cranial nerves (Dejerine's *aberrant pyramidal fibers;* see Fig. 79, c). In the pons they lie along the ventral surface of the medial lemniscus near its medial border and are easily recognized in fœtal specimens by their late myelination as compared with the lemniscus, which they join farther down. They are distributed to the nuclei of the fifth nerve, the seventh nerve and the ninth, tenth, eleventh, and twelfth cranial nerves.

The decussation of the brachia conjunctiva has been mentioned. In lower vertebrates at least (van Gehuchten) a few fibers descend after crossing. They can be traced only as low as the formatio reticularis of the bulb, and their significance is unknown. Most of the fibers of the brachia conjunctiva after crossing end in the *red nucleus;* many pass onward to the lateral nucleus of the thalamus. The *medial lemniscus* is displaced laterally by the decussating brachia conjunctiva. The *lateral lemniscus* gives fibers to the inferior colliculus and passes lateralward on its way to the medial geniculate body.

The *medial longitudinal fasciculi* of both sides meet in the middle line by a series of small bundles arranged in a ventral curve (Figs. 66 and 67). Embedded in their dorsal surface is the nucleus of the trochlear (fourth) nerve.

The mesencephalic root of the fifth nerve and its nucleus end at this level. Their connections and significance are unknown.

SECTION OF THE MESENCEPHALON AT THE LEVEL OF THE SUPERIOR COLLICULI

(Figs. 67, 68 and 23)

The basis pedunculi and substantia nigra require no special mention.

Opposite the lower part of the superior colliculi the fibers of each *brachium conjunctivum* are collected after crossing into a large rounded bundle, and here the roots of the third nerve pass freely through them (Fig. 67). A little higher (Fig. 68) the greater part of each brachium ends in the *red nucleus;* a few fibers on the medial side of the red nucleus pass on to the lateral nucleus of the thalamus in which they end.

Forel's decussation or the rubrospinal decussation. If a section at the right level be examined (Fig. 67), many new fibers are seen to spring from the medial side of the red nucleus. They form a coarse fibered

decussation between the ventral parts of the branchia conjunctiva. After crossing these fibers form the *rubrospinal tract* and assume a longitudinal direction, ventromedial to the brachium conjunctivum. At the upper end of the pons this tract lies dorsal to the medial margin of the medial lemniscus (Fig. 83). At the lower end of the pons it passes through the corpus trapezoideum lateral to the superior olive; in the oblongata it lies medial to the gelatinous substance. In the cord it lies just in front of the lateral cerebrospinal tract (Fig. 69). Its fibers end in connection with the cells of the lower motor neurons. The *rubrospinal tract* is probably the only efferent tract passing from the cerebellar hemispheres to the lower motor neurons. It is probable that cerebellar influences reach the eye muscles also through the red nucleus. The efferent tract from the vermis is probably by way of or beside the vestibulospinal tract.

The *red nucleus* receives fibers on its lateral side from the globus pallidus of the lentiform nucleus. These fibers may be seen joining it in the subthalamic region above the level of the section under consideration. Thus the rubrospinal tract is also the tract by which the *lentiform tone controlling influence* reaches the lower motor neurons. It seems more probable that the pallidal control is exerted directly over the red nucleus itself.

The *medial lemniscus* lies lateral to the red nucleus and is triangular in section. It can be seen giving collaterals to the superior colliculus. Immediately dorsal to the lateral margin of the basis pedunculi is the *medial geniculate body* (Fig. 68), a considerable mass of gray matter. Medial to it is the *lateral lemniscus;* the fibers of this tract end here in the medial geniculate body, which is the lower center for hearing. From the medial geniculate body the auditory radiation relays this tract to the superior temporal gyrus, which is the cortical center for hearing.

Fasciculus longitudinalis medialis and nuclei of the third nerve. The *medial longitudinal fasciculus* here meets that of the opposite side to form a V (Figs. 67 and 68). Within the V are the various groups of nerve cells which form the *nucleus of the third or oculomotor nerve.* The nucleus is about 5 mm. long (Figs. 12 and 13) and extends a little above the level of the superior colliculi into the lateral wall of the third ventricle. It is composed of several distinct groups of cells, one of them lying in the middle line and unpaired (Fig. 68). Collections of small cells supply the sphincter iridis and ciliary muscle, thus forming the *visceral or splanchnic efferent* nucleus of the oculomotor nerve. The nerve fila leave

the ventral surface of this in many bundles which pass through the medial longitudinal fasciculus (Figs. 68 and 23) between the red nuclei and through the upper part of the brachia conjunctiva after their decussation. The fila emerge by the medial side of the basis pedunculi. Certain fila of the third nerves intercross so as to join the nerves opposite to the nucleus from which they spring; probably they supply the medial rectus for the purpose of conjugate deviation in conjunction with the opposite external rectus muscle, which is supplied by the sixth nucleus. The coördinated action of these muscles is brought about through the medial longitudinal bundle. The nucleus of the third nerve is supplied with pramidal fibers from the opposite pramid and is connected with the opposite vestibular nucleus through the medial longitudinal bundle and with the sixth nucleus of the opposite side in the same way. It receives fibers from the superior colliculus either through the medial longitudinal bundle or through the fountain decussation, and from the corpus dentatum of the cerebellum probably through the red nucleus.

The lateral geniculate body, optic tract, and superior colliculi. A section through the upper part of the superior colliculi usually includes the lateral geniculate bodies and the ending of the optic tracts (Fig. 68). The *lateral geniculate* body (Fig. 68) is a mass of gray matter lateral to the medial geniculate body and dorsolateral to the basis pedunculi. In its ventral surface most of the fibers of the optic tract end, but some fibers go round it to reach the *superior colliculus,* while some are contributed to the pulvinar of the thalamus (Fig. 68). The superior colliculus and pulvinar also receive cortical efferent fibers from the calcarine area of the cerebral cortex. The *optic tract* (Fig. 105) derives its fibers from the homolateral side of each retina. It thus receives visual impressions from both eyes from the opposite half of the field of vision, since the light rays cross in the lens. The function of the *lateral geniculate* body is visual; its neurons form the relay to the calcarine area of the occipital lobe. A dog deprived of its occipital lobes but with the lateral geniculate bodies intact is able to avoid obstacles in walking but cannot interpret what it sees (Luciani's Physiology). Though fibers of the optic tract go to the pulvinar, destruction of the pulvinar does not cause hemianopsia. It may be that the pulvinar is associated, not with simple seeing, but with the pleasurable or painful elements in light perception. The superior colliculus does not take part in the sense of sight, but forms a reflex center coördinating eye and body movements with visual impressions. This is effected through the myelinated fibers which form its *stratum profundum.*

These arise from the gray matter of the colliculus, and partly decussate with each other dorsally (Fig. 68). Each bundle forms a graceful curve round the central gray matter (Figs. 67 and 68). It sends fibers lateralward which are lost in the formatio reticularis, and others which cross medialward through the medial longitudinal fasciculus, to which it perhaps contributes fibers. The fibers then decussate with those of the other side, crossing between the red nuclei dorsal to the rubrospinal decussation (Fig. 67) and a little higher up forming the *tecto-spinal decussation.* This is often called the *fountain decussation,* or *Meynert's decussation.* The fibers are much finer than those of the rubrospinal (Forel's) decussation. Below the decussation the fibers become longitudinal, forming the tectobulbar and tectospinal tracts.

SECTIONS THROUGH THE DIENCEPHALON

In sections through the diencephalon are found the mesencephalic structures which occupy the region ventral to the thalamus,—the hypothalamic region (see Figs. 26 and 87, a). Figure 26, stained by a myelin stain, shows that the basis pedunculi is directly continuous with the internal capsule. The relations of the peduncular fibers to the internal capsule and cerebral cortex can be established only by the study of specimens showing degeneration of the fibers. Such degeneration produced by experiments in lower mammals and the higher apes and compared with those caused by pathological lesions in man have fully demonstrated the connections of the peduncular fibers. (Compare Figs. 87, c, d and e). In this manner it has been shown that:

(*a*) All the fibers of the basis pedunculi are efferent fibers from pyramidal cells in the cerebral cortex. On the dorsal surface of the basis mesencephali, between it and the substantia nigra, many fine fibers are seen running into the substantia nigra. Of these some are collaterals from cortical efferent fibers, others are pallidonigral fibers from the globus pallidus.

(*b*) The motor area of the cerebral cortex (Figs. 90, 91 and 93; compare with Figs. 88 and 89) occupies the anterior central convolution and anterior wall of the central sulcus. Here fairly distinct areas of cortex control different groups of muscles as indicated in Figures 90 and 93; the head and neck are controlled by the lower two-fifths of the anterior central convolution, the upper extremity by the one-fifth above this, and the trunk and lower extremity by the upper two-fifths; the perineal region is controlled by an area in front of the central sulcus on the medial surface

of the hemisphere. These areas are fairly definite but are not sharply marked off from each other. The genua of the central sulcus are too irregular to have value as aids to localization. The Betz cells in this area of the cortex and their axons form the upper motor neurons.

(*c*) These upper motor neuron or pyramidal fibers are so arranged in the internal capsule (Fig. 87, e) that the cortical fibers to the cranial nerve nuclei occupy the knee of the internal capsule; the fibers for the shoulder, elbow, hand, hip, knee, and foot come behind the fibers to the cranial nerves in the order in which they are named, but the fibers to each region overlap their neighbors. Thus the knee and the thalamolentiform portion of the internal capsule contain all the voluntary motor fibers. The retro-lentiform portion of the internal capsule is purely sensory but the motor area just described is not purely motor but contains thalamocortical and corticothalamic fibers mingled with the motor fibers.

(*d*) If these voluntary fibers are traced to the basis mesencephali, they are found occupying the middle three-fifths of the basilar portion (Figs. 87, d and 79, c). Here the fibers are more mixed, but there is still a concentration of cranial nerve fibers medially, arm fibers next, and leg fibers laterally. By the time the pyramidal fibers reach the pyramid in the medulla oblongata they are so much intermingled that there is no differentiation of arm and leg fibers below the mesencephalon.

(*e*) As has been shown, by far the *greater bulk of the pyramidal fibers* cross to control the lower motor neurons of the opposite side; those which go to motor nuclei of cranial nerves cross in the plane of the individual motor nuclei for which they are destined; the spinal fibers cross mainly at the pyramidal decussation to form the lateral cerebrospinal fasciculus, a few (one-fourth of the whole, more or less) descend uncrossed in the cord as the anterior cerebrospinal fasciculus. These cross fiber by fiber till they are exhausted, usually in the upper thoracic region, though in many cases these anterior cerebrospinal fibers may be traced to the sacral segments.

(*f*) **The aberrant pyramidal fibers of Dejerine** include groups of fibers destined for the supply of cranial nerve nuclei, though not necessarily all of the pyramidal fibers which govern cranial nerves. In the mesencephalon a few small bundles lie along the lateral edge of the substantia nigra and frequently a few are scattered along the ventral border of the medial third of the basis pedunculi (Figs. 22 and 79, c). In the upper pons they appear as rounded bundles embedded in the ventral surface of the medial border of the medial fillet (lemniscus) (Figs. 79, e and 64). Lower down they

may be scattered through the medial lemniscus. They pass to cranial nerve nuclei.

(*g*) **Homolateral pyramidal supply.** A few pyramidal fibers are distributed to lower motor nuclei of the same side. Such homolateral partial supply goes to the fifth nerve for the supply of masticatory muscles, and to the seventh nerve, but only to the nuclei of this nerve which supply the muscles of the upper part of the face. The muscles of the lower part of the face have only a crossed pyramidal supply. A weak homolateral pyramidal supply goes also to the laryngeal, pharyngeal and head-turning muscles, and tongue (glossopharyngeal-vagus-accessory group and hypoglossal nuclei). The eye muscles (third, fourth and sixth cranial nerves) and lower face have only a crossed pyramidal control. In the trunk, those muscles which habitually act together—the intercostal and the abdominal muscles—and probably also the muscles which control the roots of the limbs have a weak homolateral supply; the pyramidal fibers for these run down the cord in the lateral cerebrospinal tract of the same side (Figs. 79, m and n).

(*h*) So far we have only accounted for the fibers which occupy the middle three-fifths of the basis pedunculi. The medial fifth is occupied by the *frontopontine tract*. This consists of fibers which arise in the frontal region of the cortex, descend through the anterior limb of the internal capsule (Fig. 87, e) where they are mixed with thalamofrontal and frontothalamic fibers, and pass by way of the medial one-fifth of the basis pedunculi (Fig. 87, d and 79, c) to synapse with the neurons of the nuclei pontis. The term nuclei pontis is used for all the cells of the basilar part of the pons taken together. Occasionally fibers for cranial nerve nuclei are scattered along the margin of the medial fifth of the basis pedunculi (Fig 79, c).

Similarly the lateral fifth of the basis mesencephali (or basis pedunculi) is occupied by *temporopontine fibers* (Fig. 79, c). These come from the cortex of the middle and inferior temporal gyri, reach the basis pedunculi by passing under the posterior extremity of the lentiform nucleus, and end round the nuclei pontis. The pontine nuclei relay these cortical neurons to the cortex of the opposite cerebellar hemisphere by way of the brachium pontis. The frontal area of the cortex is intimately associated with voluntary motion and the inferior and middle temporal gyri are the probable cortical centers for equilibration and vestibular tone—the cortical receptive centers for the vestibular apparatus. By means of the frontopontine and temporopontine fibers and the nuclei pontis the

cerebellar hemisphere, whose function seems to be to maintain muscular tone and coördination, is thus brought into working relations with cognate areas of the cerebral cortex.

The substantia nigra of the mesencephalon is continued a short distance into the hypothalamic region. There it receives fibers from the globus pallidus (Figs. 26 and 87, f).

The tegmentum mesencephali is directly continuous with the hypothalamic region. The *red nucleus* (Figs. 87, a and 26) and the fibers of the brachium conjunctivum medial to it terminate beneath the thalamus; degeneration experiments show that the *brachium conjunctivum* ends partly in the lateral nucleus of the thalamus and partly in the red nucleus. The decussation of the brachia is complete. The red nucleus is said also to send fibers to this nucleus of the thalamus. The balance of evidence is strongly against any of the fibers of the *medial lemniscus* going *directly* to the cerebral *cortex.*

Further connections of the thalamus. When it is stained so as to show myelin, the thalamus shows certain prominent divisions (compare Figs. 87, a and b, 26 and 168, a).

(*a*) A well-marked strand of myelinated fibers ascends from the cells of the corpus mammillare to enclose the anterior tubercle of the thalamus as in a capsule. This is the *mammillothalamic* bundle, and the tubercle is the *anterior nucleus of the thalamus.* Its function is olfactory. It is probable that in this anterior thalamic nucleus olfactory stimuli reach consciousness and give rise to impressions of comfort or discomfort, appetite or reflex nausea.

(*b*) The posterior tubercle of the thalamus or *pulvinar* (Fig. 68) is one of the end stations of the optic tract, and is probably associated with the pleasurable and disagreeable elements in light perception. The lower end station for sight as such is the *lateral geniculate body* (Fig. 68) where elementary light sensations reach consciousness. The lateral geniculate body and the pulvinar are connected by way of the optic radiation (Fig. 87, b) with the calcarine area of the occipital lobe by corticipetal and corticifugal fibers.

(*c*) The *medial geniculate body* (Fig. 68) may be considered a part of the thalamus; in it ends the lateral lemniscus from the cochlear nuclei. On its way to the medial geniculate body, the lateral lemniscus sends fibers or collaterals to the posterior colliculus (Fig. 68). These two nuclei, the medial geniculate body and the posterior colliculus, are connected with the superior temporal gyrus by centripetal and centrifugal fibers.

The medial geniculate body is the lower auditory center; here auditory stimuli reach consciousness for simple light perception.

(*d*) The *ganglion habenulæ* is connected with the striæ medullares, with the hippocampal gyrus, with a nucleus near the optic chiasm, and by the fasciculus retroflexus with the interpeduncular ganglion. Its function is olfactory and though of great interest phylogenetically, it has no clinical importance at the present time.

(*e*) In Pal-Weigert or iron hæmatoxylin specimens it may be seen that the remaining part of the thalamus, including most of its bulk, is plainly divisible into a medial nucleus which is very poor in myelinated fibers and a lateral nucleus which is very rich in such fibers (Fig. 87, a and b). In the ventral portion of the lateral nucleus end the tracts for all forms of ordinary body sense. From the ventral part of the lateral nucleus a short relay takes place to the *dorsal part* of the lateral nucleus where there is a redistribution of the different forms of sensation. Thus in the ventrolateral part of the thalamus, where all sensory tracts end in comparative concentration of fibers, a lesion will cause complete loss of all forms of sensation for the opposite side of the body, but in the dorsal part of the lateral nucleus a limited lesion is characterized by loss of one or more of the different forms of bodily sensation in very varying degree. From one or both of the lateral thalamic nuclei relays for touch, pain, heat and cold and perhaps position pass to what Head and Holmes call the *essential thalamic organ* where these sensations reach consciousness and become associated with pleasant or unpleasant feelings. This essential organ of the thalamus is probably the medial nucleus. In specimens stained for myelin (Fig. 26) this nucleus is seen to be very poor in myelinated fibers; but with pyridin silver and Golgi silver stains it is seen to be very rich in Golgi type II cells and in unmyelinated network. The thalamus is connected with the cerebral cortex, both in the frontal and parietal lobes by *thalamocortical* fibers so that sensory memories may be stored and compared in the cortex, and by *corticothalamic* fibers whose function appears to be to control the tendency to over-reaction of the thalamic organ. Thus excessive reaction to conscious stimuli may be kept under voluntary restraint (Head and Holmes, *Lancet*, 1912).

The *lateral nucleus of the thalamus* is also a relay station to the anterior central gyrus and to the posterior central and superior and inferior parietal convolutions; it relays all forms of body sensation underlying coördinated motion. These fibers occupy the internal capsule where they are intermingled with fibers of the upper motor neuron. All the thalamo-

cortical connections together form the thalamic radiation. By means of this the afferent impulses which underlie voluntary activities are conveyed to the motor cortex. By way of this radiation stimuli are carried to the posterior central and parietal convolutions and in these gyri tactile and muscle sensations, sense of weight, vibration, and texture are referred to the parts of the body from which they spring (tactile localization or topognosis), and are registered, stored as memories, and compared with previous sensations of a similar character. It is believed that the *cortical centers for touch and the sense of posture* are in the posterior central convolution, and that the *center for the recognition of form* by touch and perhaps the sense of relative weight are in the neighborhood of the intraparietal sulcus (Fig. 142). The intimate connection of these centers by association fibers (Fig. 149) with all parts of the cortex, including motor, sensory, and association areas, provides for the sum of activities which underlies conscious being. It is probable that the lemniscus fibers which end in the ventral part of the lateral thalamic nucleus, where all forms of bodily sense are somewhat concentrated, undergo first a short relay to the dorsal part of the lateral nucleus where the various forms of sensation are segregated, and that the relays to the medial thalamic nucleus and cortex are from this dorsal part of the lateral thalamic nucleus. The cortical center for judgments of more or lessness appears to lie along the upper lip of the lateral sulcus; and injury to the anterior central gyrus is always associated with loss of postural sense (Fig. 142).

The corpus striatum and its connections (Fig. 87). The corpus striatum is phylogenetically and probably physiologically divisible into two organs, the palæostriatum or ancient striate body, and the neostriatum or new striate body.

The palæostriatum, which is represented in mammalia by the globus pallidus, is found in all vertebrate brains from those of the shark family upward. In sharks and bony fishes it is represented by the basal ganglion and is the controlling motor organ as there is no motor cortex. Its cells are large and multipolar, of the efferent projection nueron type; it is rich in myelinated fibers. In higher mammalia its function is subordinate in proportion as the voluntary motor cortex is developed. Evidence is accumulating which seems to indicate that in man the palæostriatum exerts a steadying influence on the lower motor neuron (S. A. K. Wilson, *Brain,* 1911-'12) and through this on muscular action and so counteracts

the tendency to overaction of the tonic mechanism on the lower motor neuron. This it does through fibers relayed in the red nucleus to the opposite rubrospinal tract (Fig. 87, f). Its connection with the red nucleus, nucleus hypothalamicus (of Luys) and substantia nigra form the ansa lenticularis. There is probably an efferent tract from the cells of the substantia nigra the fibers of which are mixed with the pyramidal tract. The globus pallidus may also have a steadying action on cortical motor activities indirectly through its pallido-thalamic fibers. Disease of the globus pallidus results in general muscular rigidity of the opposite side of the body with coarse tremors.

The neostriatum consists in mammals of the caudate nucleus, the amygdala and putamen; it appears first in lizards and is of large size in birds, in which the voluntary motor cortex is absent. In birds and the lower mammals the caudate nucleus and putamen are one continuous mass, but in higher mammals and especially in man they are almost completely separated by the internal capsule. These two parts of the neostriatum are similar in structure. They have a few large multipolar cells but are rich in small cells of the Golgi type II character. They are poor in myelinated fibers, but have an exceedingly complicated nonmyelinated network. Fibers pass from the thalamus to the caudate nucleus and thus there is provided an afferent tract for the motor activities of the striate body. The caudate nucleus is connected with the putamen and the putamen sends fibers to the globus pallidus. Mme. Wogt (Journal. f. Psych. U. Neur., 1912) attributes the involuntary movements of chorea and athetosis to disease of the neostriatum, and Hunt (*Brain,* 1917) considers it capable of controlling automatic movements. The writer wonders whether those motor activities which by long practice become almost automatic, such as typing, stenography, and piano-playing, may be as it were handed over to the corpora striata, thus relieving the cerebral cortex. It is known that a dog deprived by Goltz of its cerebral cortex, but having its basal ganglia and thalamus comparatively intact, when it had recovered from the shock of the repeated operations necessary to effect this, showed no visible loss of motor power. It moved about incessantly except when asleep, fed and maintained fair health, but had lost all memory, will and other signs of individuality, and was apparently a mere reflex mechanism. (See Schaefer's Physiology; so also Luciani's dogs, Luciani's Physiology). Goltz's dog had its temporal lobe left intact so as to avoid injuring its optic tracts.

MICROSCOPIC STUDY OF ADULT SECTIONS OF CORD AND BRAIN STEM

The description which follows is intended for use as a laboratory guide for the study of actual sections.

The specimens described are stained by the Weigert or iron hæmatoxylin technique for myelin. This stains myelin so that in cross-sections viewed by the naked eye or under a magnifying lens, white substance shows as a series of large or small circles; the edges of these in thin sections are much darker than the centers. Usually the axons do not stain. In longitudinal presentation the myelinated fibers show as irregular dark streaks. The neuroglia network in iron hæmatoxylin specimens shows as yellowish or gray fibrillar substance which forms a continuous lining inside the pia and sends strands of glia fibrils inward between the bundles of nerve fibers. The pia invades the white matter only as sheaths for the blood vessels like the connective tissue trabeculæ in other organs. In iron hæmatoxylin specimens nerve cells stain pale gray, the tigroid substance is darker, the nucleus is a gray circle, and the nucleolus, a dark dot. Neuroglia cells show only the very dark nuclei which almost fill the cell. Red blood cells show as black bodies in rows in capillaries or in groups in larger vessels. Leucocytes show nuclear capillary networks. Pal-Weigert specimens show only myelin unless counterstained. Usually Upson's Carmine or alum carmine is used as the cell stain. Sections of brain stem cut in paraffin and stained with either Pal-Weigert or iron hæmatoxylin show the axons stained black and surrounded by very thin dark rings of myelin.

General directions: For the further study of sections of the cord and brain stem an aplanat lens may be used on a simple stand giving a field of 15 mm. with magnification of 6 diameters, and a microscope with long ocular which with a Spencer Lens Company's 32 mm. objective gives a field of 3 mm. with magnification of 10 diameters. A long ocular with a Bausch & Lomb 32 mm. objective gives a field of 5 mm. and a magnification of about 20 diameters. The higher power provided in the laboratory is a Spencer Lens Company's 16 mm. objective, which in this combination gives a field of 2 mm. and a magnification of about 40 diameters. For detailed examination of cell groups a short ocular and 4 mm. objective may be used. In all cases low power ($\times$ 10) should be used for orientation, and higher power ($\times$ 40) for details.

SPECIMEN I. Adult lumbar cord. Pal-Weigert stain (compare with Figs. 4 and 5).

Pia. The pia is very thin; it is fibrous in character and blood vessels are imbedded in it. It forms a distinct anterior median septum. Occasionally vessels are seen to penetrate the cord, carrying with them delicate pial sheaths and surrounded by perivascular lymph spaces. In the gray matter the capillaries are very abundant and are seen as strings of erythrocytes, almost black with this stain.

Neuroglia shows as a pale yellow ground substance forming strands in the white matter, in which the myelinated bundles are embedded. A rather distinct posterior median septum of neuroglia is evident. If alum carmine has been used as a counterstain the neuroglia takes a delicate pink stain. If the section catches a blood vessel running into the posterior median septum it will appear as if there is a posterior median fissure occupied by pia mater.

WHITE MATTER

Posterior nerve roots here enter in single bundles (Fig. 4) and the very largest appear to enter the gelatinous substance directly. Most of the larger myelinated fibers of the posterior nerve roots enter the posterior white columns medial to the posterolateral column and almost immediately become longitudinal. This area is therefore well called the posterior root zone (Fig. 69). The great bulk of the fibers becomes longitudinal (compare Figs. 4, 69 and 74). Figs. 74 and 75 show that on entering the cord each entering fiber splits into an ascending and a descending branch and gives collaterals and terminals at many levels to the gray matter. These collaterals to the gray matter are seen entering from the posterior white columns and form longitudinal bundles in the gelatinous substance and medial side of the gray column.

Anterior nerve roots are seen to leave the anterior surface of the gray column in two or three or five or six bundles of fibers, varying according to the chance variation in the position of the section. The bundles are composed of converging fibers formed of the axons of the motor nerve cells in the anterior gray columns. Where the nerves of the cauda equina are given off the anterior nerve roots may take a longitudinal direction just under the pia.

White columns. The posterior columns. There is no differentiation of the posterior column at this level into a fasciculus gracilis and fasciculus cuneatus. From the posterior columns large numbers of nerve fibers are seen to enter the medial surface of the posterior gray column, either ending there or passing into the anterior gray column.

The posterolateral column (Column of Lissauer). Between the gelatinous substance and the periphery, on either side, but especially ventrolateral to the posterior nerve root, the posterolateral column is distinguishable by the delicacy of its myelinated fibers. Under higher powers with Golgi silver or pyridin silver stains most of the fibers of this column are seen to be naked axons. The nonmyelinated fibers of this column have been proved by Ranson to convey (in the cat) impressions of pain, and probably also heat and cold. The writer thinks that the fine myelinated fibers of this column may convey delicate touch (epicritic touch). Both sets of fibers enter the gelatinous substance synapsing ultimately with tract cells in the posterior gray columns, either directly or through the intermediation of Golgi type II cells in the gelatinous substance.

The fibers entering the medial surface of the posterior gray column probably convey impressions of touch and muscle sense, and synapse with tract neurons to the cerebellum and other tract-forming cells or with anterior horn cells for cord reflexes (compare Figs. 4 and 69).

Anterolateral white column. This includes all the white matter in front of the posterolateral column. It is not divisible into an anterior and a lateral column, as the anterior nerve roots are unlike the posterior nerve roots in that they do not emerge from a distinct groove in a single row; nor are the tracts which are physiologically differentiated in any way limited by the emerging anterior nerve roots. This may be seen on looking at Figure 69. Under higher powers the axons and myelin sheaths show great difference in size, and with pyridin silver stains, many nonmyelinated fibers are found between the myelinated ones. Cajal believes that these nonmyelinated fibers are either endogenous fibers from the gray matter that have not yet acquired myelin sheaths, or collaterals or terminals about to enter the gray matter that have lost the myelin sheaths.

White commissure. The white commissure is seen to be a decussation. It consists of fibers passing from the anterior white column to the opposite gray column.

Gray columns. Under low powers the anterior nerve roots are distinguished by the large size of the axons in axon stains and by the large myelin sheaths in myelin stains. They are seen to converge from all directions toward the anterior root bundles. Bundles of myelinated fibers enter the gray matter from the posterior white columns. Many end in the posterior gray column; others reach the anterior gray columns. They are finer than the anterior nerve roots. With axon stains many non-

myelinated fibers are seen running in all directions and bundles of them pass into or out of the lateral columns all round the periphery of the gray column. This is especially marked in pyridin silver staining where dendrites also are stained, adding greatly to the complexity of the network.

Posterior gray column. The *cap of gelatinous substance* is well marked, and crescent shaped. Its cells cannot be made out under low powers. Many bundles of large fibers pass longitudinally through this gray column ventral to the gelatinous substance on its medial side, and very many vertically directed fine fibers are scattered among the coarser bundles and more laterally form a steel gray coating beneath this gelatinous substance. They form a fibrillar network round the cells of the posterior gray column. (Under higher powers and with suitable stains these regions are seen to be rich in nerve cells.)

Anterior gray column. Each cell lies in a space which is probably a lymph space artificially enlarged by the process of hardening and dehydrating. Note the grouping of the cells and compare it with Figure 8. The medial group consists of small cells and supplies muscles close to the vertebral column. The lateral group of cells consists of larger cells and supplies the muscles farthest removed from the vertebral column for this particular segment of the cord.

The central canal in most adult specimens is full of cast-off degenerated neuroglia cells of the ependymal cell type.

The gray commissure is seen to consist of a small number of delicate decussating fibers. Only a few are myelinated so that most of the fibers of the gray commissure can be seen only with axon stains. They pass from cells of the posterior gray column to the white columns of the same and of the opposite side and are probably axons of association neurons or tract axons for pain, heat and cold.

No other cell groups can be made out under these powers.

Note the wealth of blood capillaries in the gray matter seen as strings of erythrocytes stained almost black, and the perivascular lymph spaces much enlarged by the method of preparation.

SPECIMEN II. (Compare Figs. 3 and 7.)

Section 2 is from the *lower thoracic region of the cord,* cut in celloidin and stained by the Pal-Weigert method with Upson's carmine or alum carmine as a counterstain. It shows a typical myelin stain.

The subpial neuroglia with its delicate processes entering the white substance is well seen in these sections.

White columns. There is no subdivision of the dorsal white column at this level. The dorsolateral column is again marked by the small size of its myelinated fibers. It is very small, as may be expected from the small cutaneous distribution of the thoracic nerves. For a similar reason the number of nerve fibers entering the gray substance from the posterior column is small.

Gray matter. The characteristic spear-head shape of the gelatinous substance in this region shows well. The small amount of gelatinous substance is in proportion to the small skin area supplied by a thoracic nerve.

Anterior gray column. The *medial group of motor neuron cells* is the best marked. It supplies spinal muscles. A few *more laterally* placed and perhaps a *central* group supply the muscles of the abdominal or thoracic wall. There is a well-marked *intermediolateral cell column* occupying a distinct lateral horn. (Compare with Fig. 7.) The cells are smaller than anterior column cells. This is the nucleus of the spinal efferent neurons to the sympathetic ganglia. A prominent group of cells in this section is the group that forms the *nucleus dorsalis* (Clarke's Column). They are large multipolar nerve cells, nearly as large as lower motor neuron cells, are ovoid or pyriform in shape and show a large nucleus and nucleolus and tigroid substance. Fibers from the posterior nerve roots are seen to come into relation with them from the posterior white column. Their axons form the posterior (dorsal) spinocerebellar tract (Fig. 69). This nucleus extends from the second thoracic to the first lumbar segments inclusive. The *central canal* in this specimen also is usually filled with degenerated ependymal cells.

SPECIMEN III. (Compare with Figs. 2 and 6.)

About the middle of the cervical enlargement.

The posterior funiculus is here differentiated into a well-marked *fasciculus gracilis* medially, separated from the *fasciculus cuneatus* by a septum of neuroglia. The fasciculus gracilis here shows that its fibers are uniformly smaller than are the bulk of the fibers of the fasciculus cuneatus. The fibers of the posterior column destined for the nucleus gracilis and nucleus cuneatus are smaller than those which end in the gray matter of the cord. There are very few unmyelinated fibers in the posterior columns.

The anterolateral white column needs no special mention except for two points which can be made out under a high power. Just lateral to the neck of the posterior gray column is the formatio reticularis in the gray strands of which can be found an occasional nerve cell. Along the

surface of this column for some distance, commencing outside the posterolateral column, the myelinated fibers are uniformly large. This is the posterior spinocerebellar tract (Fig. 69).

The posterolateral column is as before marked by the small size of its myelinated fibers. Pyridin silver staining would show that it is very rich in nonmyelinated fibers. Medial to the posterolateral column an entering posterior nerve root may be seen. A few of the fibers, or rather collaterals from them, enter the gelatinous substance directly. The nerve root assumes a vertical course partly in the posterolateral column and partly in the gelatinous substance. Many collaterals can be seen piercing the gray matter from the longitudinal fibers. Many bundles of fine and coarse myelinated fibers, and many unmyelinated fibers run longitudinally beneath the gelatinous substance.

Gray substance. The gelatinous substance is abundant in correspondence with the large skin area supplied by this segment of the cord. Under a 4 mm. objective it shows numerous small cells, and in the neck of the posterior gray column are a few larger cells.

Anterior gray column. Compare the grouping of the larger lower motor neuron cells with Figure 6. In the anteromedial group the cells are scanty and comparatively small. There are several lateral groups of cells and these cells are large. They supply the deltoid, triceps, biceps, brachialis, and brachioradialis. The axons of these lower motor neuron cells leave the medial portion of the ventral surface of the column in four to six bundles (Fig. 2).

A few rather large cells may be seen in a position corresponding to that of the nucleus dorsalis in the thoracic cord. Investigations by McNalty and Horsley (*Brain*, 1909) indicate that their axons go to the homolateral spinocerebellar tracts. Many fine myelinated and unmyelinated fibers form an intricate network in the anterior gray column.

SPECIMEN IV.

The first cervical segment (compare with Fig. 1).

The pia and the neuroglia appear as in the preceding sections. The *fasciculus gracilis* is as before separated from the *fasciculus cuneatus* by a septum of neuroglia. The fasciculus gracilis here forms a distinct prominence on the dorsal surface of the cord. The *posterolateral fasciculus* is again characterized by the small size of its myelinated fibers; these are mixed with many nonmyelinated fibers which are usually invisble in sections stained by iron-hæmatoxylin or Pal-Weigert hæmatoxylin. It is here mainly formed by the *spinal tract of the fifth cranial nerve* and asso-

ciated with it is a considerable enlargement of the gelatinous substance. At the bottom of the anterior longitudinal fissure is seen the lower end of the *pyramidal decussation.* Some of the pyramidal fibers after crossing may be seen passing through the neck of the anterior gray column to join a series of bundles of fibers which lie in a lateral recess between the posterior and anterior gray columns (Fig. 1). The white fibers here are separated into distinct bundles by a reticulum of neuroglia containing nonmyelinated nerve fibers and occasional nerve cells. The appearance so produced has led to the name of the *reticular formation* of the spinal cord which designates this area. The bundles of nerve fibers seen in cross section are mainly the newly grouped fibers of the *lateral cerebrospinal fasciculus,* formed by the decussation of the greater part of the pyramid immediately above this level (compare with Figs. 56 and 57).

The cells of the lower motor neurons in this section are difficult to see under the low powers employed and with this stain. They are pale yellow bodies the color of the neuroglia, each with a small blackish central nucleolus, and each cell surrounded by a clear space, or in sections counterstained with carmine they are pink with a red nucleolus. They are the motor cells of the first cervical nerve. If the anterior gray column is examined more closely, a ventromedial, ventrolateral, and dorsolateral group may be distinguished. The dorsolateral group is part of the *nucleus* of the *eleventh cranial nerve* (Fig. 56). The *central canal* is usually filled with degenerated ependymal cells. There may be many vessels full of blood cells, and as in other sections examined, these are especially abundant in the anterior gray columns.

From our studies so far it is evident that no functional differentiation of tracts in the white columns of the cord and in the brain stem is possible by this method of staining in normal adult cords. They are differentiated mainly by the examination of degenerations due to experimental lesions in lower mammals and apes, and pathological lesions in man. Recent degenerative changes are revealed by the Marchi method (see introduction) where the degenerated myelin is stained black by osmic acid after hardening in chrome salts; while later degenerative changes are made manifest by the failure of degenerated myelin to stain by an iron hæmatoxylin or Pal-Weigert myelin stain (contrast Fig. 81 with Fig. 82).

THE NERVE TRACTS STUDIED BY DEGENERATIONS

Much may be learned by studying experimental degenerations in animals by the Marchi method. If nerve cells from which axons spring be

destroyed or axons be cut off from their nerve cells, the axons degenerate. After an interval of two to three weeks (not more or less), the specimens secured may be stained by the Marchi technique, and very accurate information may be obtained by observing the fatty degeneration of the myelinated fibers involved. This method, however, is not applicable to nonmyelinated fibers and little is known of their connections.

In the following study it has been thought better to use cases available in man, and only occasional reference is made to experiments in animals; but the two classes of observations correspond very closely in their results. Only one or two sections will be shown that the student may understand the appearance of such sections. The rest of this description must be studied with the aid of diagrams.

The upper motor neuron as shown by the degeneration method. In Figures 87, c and d, the dotted areas show the degenerated tracts in the internal capsule and mesencephalon of a monkey after destruction of the whole motor cortex of the right hemisphere. The limitations for cortical areas of the face, arm, and leg in the internal capsule are estimated from results of experiments destroying cortical areas which were shown by previous stimulation to control these groups of muscles. In the internal capsule it is to be noted that the degenerated fibers occupy the knee and lenticulothalamic portion of the posterior limb of the capsule; face, arm, and leg areas overlap each other; the cranial nerve area occupies the knee. The degenerated fibers are more concentrated near the lentiform nucleus. The figure on the right side (87, e) shows these areas as they have been demonstrated in the internal capsule in man by repeated examinations of degeneration occurring in cases of softening involving localized areas of the motor cortex. Figure 87, d shows the degenerated area in the basis pedunculi in a monkey. In man it occupies the middle three-fifths of the basis pedunculi as will be seen later.

Figures 79, a to n, show well the tracts which degenerate in man when the bulk of the fibers from the motor area together with a considerable part of the motor cortical cells have been destroyed by occlusion of branches of the middle cerebral artery. In Figures a and b the dotted areas indicate the extent of the lesion. The superficial cortical softening has affected little more than the head area of the cortex (compare with Fig. 90), but the deep softening as seen in Figure b has interrupted pyramidal fibers from nearly the whole of the motor area. Figure c through the mesencephalon shows degeneration in the middle third of the basis mesencephali, encroaching on the outer third more than was to be

expected. There is also shown a narrow strip of degenerated fibers along the medial ventral border of the basis pedunculi which belongs to Dejerine's aberrant pyramidal fibers destined for the motor nuclei of cranial nerves; and there are two small rounded bundles of pyramidal fibers just lateral to the substantia nigra which are also to be classed with Dejerine's aberrant fibers and are also destined for the motor nuclei of cranial nerves.

Figures 79, d, e, f, g show the degenerated areas in the basilar portion of the pons and these figures show also small pyramidal bundles in the medial lemniscus, which represent the downward continuation of Dejerine's aberrant fibers. These have been called the accessory fillet, a name so misleading that it should not be used. Figures h and i through the medulla oblongata show the concentration of the motor tract in the pyramid, but also show some fibers separating off laterally. These eventually form the homolateral pyramidal fibers and are destined for the pyramidal supply of homolateral muscles which act habitually in synchronism with muscles of the other side; such muscles are the intercostal, abdominal and perineal muscles, and possibly also hip and shoulder muscles. The fifth, seventh, ninth, tenth, eleventh, and twelfth cranial nerves also have a homolateral pyramidal supply, but this is probably by way of the aberrant pyramidal fibers of Dejerine just described.

Figure 79, j, shows the decussation of the greater part of the pyramid and Figure 79, k, taken through the third cervical segment, shows the fully formed lateral cerebrospinal tract, the ventral cerebrospinal tract not yet crossed, and homolateral fibers which are partly scattered through the area occupied by the lateral cerebrospinal tract from the right hemisphere, and partly in the periphery of the anterolateral column.

In the cervical segments, of which the third and the eighth are figured (Figs. 79, k and l) the lateral cerebrospinal tract is separated from the surface of the cord by an undegenerated area which is occupied by the dorsal spinocerebellar tract; lower down the lateral cerebrospinal tract reaches the surface, though this is not apparent in these figures, probably because the fibers from the leg area of the cortex were not completely interrupted by the lesion. The ventral cerebrospinal tract in this case extends downward even into the sacral segments, and there are uncrossed fibers in the lateral columns of the cord as low as the third sacral segment; probably these supply the sphincters and the perineal muscles.

The lower motor neuron. Sections of the cord and brain stem (Fig. 78) from a patient dying three weeks after the onset of an attack of anterior poliomyelitis, show the effect of the destruction of lower

motor neuron cells by this disease. In this particular case the disease affected chiefly the lumbar segments, but also involved the cervical enlargement and smaller areas higher up. The section through a sacral segment (Fig. 78, 1) and one from the cervical enlargement (Fig. 78, 3) show by cross shading the diseased areas of anterior column cells; in these segments the anterior nerve roots may be followed as they emerge from the cord, and the degeneration consequent upon the destruction of their nerve cells is easily seen. Part of the nucleus of the accessory nerve was also destroyed (Fig. 78, 4 and 5) and the sections through the second cervical segment and through the pyramidal decussation show the course of the spinal portion of the root of this nerve by rows of dots representing degenerated fibers.

Experimentally if motor nerve roots be avulsed from the cord or brain stem, and forty-five days allowed to elapse before the animal is killed, a retrograde fatty degeneration can be shown in the submerged portions of the avulsed roots, together with chromatolysis of the cells from which they arose. Van Gehuchten has used this method extensively to demonstrate the course of lower motor neurons both cranial and spinal. He has shown that with the exception of the fourth cranial nerve and a few fibers of the third cranial nerve, no lower motor neurons cross the median plane, and he has furnished beautiful pictures of the origin of the various motor cranial nerves.

The lowest sensory neuron may also be studied by degeneration methods. Figure 80 affords an example of one of those rare cases in a human subject in which the disease is so accurately localized that the lesion amounts to a physiological experiment. The sections were taken from the cord of a patient dying with carcinoma of the spinal column; the third right lumbar nerve got caught in the tumor; no other nervous lesion existed. In Figure 80, e, the degenerated posterior nerve root is seen entering the posterior and posterolateral columns. Some collaterals enter the posterior gray column at once. Other fibers are seen to ascend and descend in the posterior column, at first close to the gray column; both the ascending and descending fibers diminish rapidly and both tend to approach more closely the median septum the farther they travel.

Taking the ascending fibers first, there is a gradual shifting medialward till in the first thoracic segment (Fig. 80, b) they occupy a narrow strip close to the posterior median septum, forming part of the fasciculus gracilis; they end on the dorsal surface of the nucleus gracilis where they are relayed.

Passing now to the descending fibers in the fourth lumbar segment immediately below the diseased entering root (Fig. f), one finds that there are 500 dots in this section; at the third sacral segment (Fig. h) there are only 85 dots, showing that there is a rapid diminution of the descending fibers. At first these fibers are grouped along the medial side of the gray column (Fig. f), but in the first sacral segment they form a scattered strip in the region known above as the fasciculus interfascicularis or comma tract. Lower they form a strip along the median septum sometimes called the septomarginal bundle, and lower down still they occupy the "sacral triangle," a narrow strip between the septum and the posterior surface. Of course this study by Marchi's stain accounts only for the myelinated fibers of the posterior roots; the unmyelinated fibers do not yield themselves to this method of research.

Extensive investigations have been carried out on dogs, cats, and monkeys, in which the posterior nerve roots in different regions have been cut. The observations made are in general agreement with the arrangement outlined; it can also be shown that in the fasciculus gracilis layers of long fibers from the sacral, lumbar, and lower thoracic regions are so arranged that the most medial fibers are those that have traveled the greatest distance. It has also been demonstrated that fibers from the posterior nerve roots of the cervicobrachial region of the cord descend to the lumbo-sacral enlargement, and that fibers from the lumbo-sacral nerve roots ascend to the cervico-brachial enlargement, thus providing for coördinated reactions of upper and lower limbs.

Endogenous spinal neurons by the degeneration method. Endogenous spinal neurons are neurons whose cell bodies are in the spinal gray matter. They may be classified as follows (see Fig. 69):

1. *Lower motor neurons.* In these the cells are in the anterior gray columns and the axons form the anterior nerve roots. The degeneration of these has been described.

2. *Tract forming neurons,* including:

(*a*) The large cells of the nucleus dorsalis whose axons form the dorsal spinocerebellar tract, relaying muscle sense to the cerebellum.

(*b*) Cells in locations still undetermined within the posterior gray columns whose axons form the ventral spinocerebellar tracts partly homolateral and partly crossed. These also relay muscle sense to the cerebellum.

(*c*) Cells of the posterior gray columns whose axons form the opposite posterior spinothalamic tracts; the decussation of these takes place either

in the ventral white commissure or more likely in the gray commissure. These neurons relay pain, heat, and cold to the thalamus.

(*d*) Cells of the posterior gray columns whose axons form the opposite anterior spinothalamic tracts. These also decussate either in the anterior commissure or in the gray commissure. They relay simple touch and pressure to the thalamus.

(*e*) Cells probably in the anterior columns whose axons form the spinotectal and spino-olivary tracts. These relay muscle sense to the superior colliculi and to the inferior olives respectively. They are probably uncrossed.

3. *Spinal association neurons.* These are cells in various situations in the gray matter whose axons travel short distances up and down, forming the fasciculi proprii of the spinal cord and sending collaterals and terminals back into the spinal gray matter. Their function is to link up different levels of the spinal cord forming an important part of the mechanism underlying spinal reflexes (Fig. 74, b; see also Fig 76 of Sherrington's reflex dog).

Only the descending association neurons can be demonstrated experimentally. If the spinal cord of a dog be cut in the upper thoracic region and the dog be kept alive for approximately a year, all trace of the fatty matter resulting from the consequent degeneration will be removed by absorption. If after this interval the cord be again cut several segments below the original section, and three weeks allowed to elapse before killing the dog, the cord below the second section will show extensive degeneration in the fasciculi proprii. This indicates the degenerated axons of association neurons whose cell bodies lie in the segments between the upper and lower sections and whose axons descend to more caudal segments of the cord.

The endogenous neurons of the cord as illustrated by cases dying of acute anterior poliomyelitis. The Figures 78, 1 to 8, are taken from an important article by Batten & Holmes (*Brain,* 1913), which throws light on this subject. They are selected from a series of patients dying of acute poliomyelitis at such times as permitted staining of the cords by the Marchi technique. Poliomyelitis destroys the gray matter extensively, but the posterior gray columns for the most part escape. In the figures shown the cross shading in the sections through a sacral and through a cervical segment show the chief areas of cell destruction. In these segments, as has been pointed out, emerging anterior nerve roots are found degenerated; but in the other segments the ascending degen-

erated fibers indicate the course of ascending endogenous neurons. The spinocerebellar tracts, both anterior and posterior, have suffered considerably from the destruction of their cells in the gray matter and their position in the cord and oblongata is indicated by the stained degenerated myelin. The shifting of the posterior spinocerebellar tract backward into the restiform body and the ending of this in the vermis is well seen in the sections of the medulla oblongata and pons, and the position of the ventral spinocerebellar tract in the pons is demonstrated. There is a general rapid diminution in the number of degenerated fibers as one ascends above a focus of destruction, showing that short association neurons are numerous; and the usual tendency of the longer neurons to assume a peripheral position as they ascend is evident. Many of the longer endogenous nuerons end on the dorsal surface of the inferior olive, thus indicating a spino-olivary fasciculus. Numerous endogenous neurons cross in the ventral commissure (decussation). In the posterior columns the only degenerated fibers are close to the medial side of the posterior gray columns, and thus it seems to be demonstrated that the fasciculus interfascicularis, septomarginal bundle, and sacral triangle consist of descending root fibers and do not contain endogenous fibers. The cornucommissural tract, however, consists of endogenous axons.

The section through the pons shows bilateral destruction of Deiters' nucleus by the disease, and the degenerated fibers flowing from it show how its axons contribute largely to the formation of the medial longitudinal bundles and vestibulospinal tract.

The rubrospinal tracts and tectospinal tracts have been demonstrated in monkeys and lower vertebrates by study of the degeneration following experimental destruction of the corpora quadrigemina and of the red nuclei, and these observations have been found to hold very closely for man as shown by the degenerated areas found in disease of the mesencephalon. Collier and Buzzard's observations on the rubrospinal tracts are illustrated in Figure 83.

The tracts from Deiters' nucleus are described as they have been experimentally demonstrated by Fraser (Jour. Phys., 1901-'02), from whose article Figures 77, 1 to 6, are copied. The section from the lower pons (Fig. 77, 1) shows the destruction by galvano-cautery of the left Deiters' nucleus. The degenerations figured at the level of the lesion and above it show that its ascending axons largely form the medial longitudinal bundle of the opposite side, contributing a few fibers to the medial longitudinal bundle of the same side; while the degenerations figured at

the level of the lesion and below it show that fibers descend from Deiters' nucleus to form the vestibulospinal tract, chiefly on its own side but partly crossing to the opposite side, and that the medial longitudinal bundle of its own side is the one which receives most of this series of descending fibers. It is further shown that the medial longitudinal bundle and vestibulospinal tract are continued downward in the anterior column of the cord (Fig. 77, 6 and 7). The fibers from Deiters' nucleus to the roof nuclei of the cerebellum are also well seen at the level of the lesion. This series of figures (77) should be compared with Fig. 78, 7 and 8 showing the degenerations resulting from disease of Deiters' nucleus. In this series the lesion is multiple, so that the fibers from Deiters' nucleus have to be carefully differentiated from other tracts involved.

Thus it is shown that Deiters' nucleus, one of the terminal nuclei of the vestibular nerve, is connected with the nuclei of the eye muscles of both sides, but especially with those of the opposite side by fibers which reach them through the medial longitudinal bundles; and that it is connected with the lower motor neurons in the spinal cord by two routes, viz.: the vestibulospinal tracts and mediäl longitudinal bundles, but this connection is mainly homolateral throughout and apparently entirely so in the lumbosacral region.

Degenerations in complete transverse lesion of the spinal cord. After having studied the degenerations of separate tracts, one is prepared to understand the degenerations found *above* and *below* the lesion in a case of complete transverse lesion of the spinal cord in man. This is illustrated by figures (Fig. 81) from a case described by Collier and Buzzard (*Brain,* 1903). In all essentials they agree with the results obtained in our own cases. The lesion is of the seventh thoracic segment and figure *q* of the series is drawn from a section in its immediate neighborhood. Here the degenerated fibers are scattered everywhere and are due to the local traumatism, but at a distance of one or two segments above and below the lesion the degenerations form very definite patterns. The fibers which degenerate below the lesion are the axons of cells higher up, and the fibers degenerating above the lesion are of course axons of cells below the lesion; both have been cut off from their cell bodies.

The degenerations *below the lesion* (so-called descending degenerations) may be considered first. Figure r, one or two segments below the lesion, shows well-marked degeneration in the areas occupied by the lateral cerebrospinal fasciculi and in the rubrospinal tracts just in front of this.

These tracts show well as gradually diminishing triangular fields in the lateral white columns; they may be traced all the way down, even in the upper part of the conus (S. 4, Fig. w). In Figures s and t, fibers from the lateral cerebrospinal tracts are seen to enter the gray matter apparently to synapse with cells in the neighborhood of the area occupied by the nucleus dorsalis. Sections from one of our own cases of injury at the seventh cervical segment show a similar penetration of the gray matter directed toward this area, but only in the lower thoracic and upper lumbar region. The exact significance of this must still be regarded as unsettled. It is believed by many neurologists, notably E. A. Schaefer, that the pyramidal fibers do not synapse directly with the lower motor neuron cells but with intercalated cells somewhere in the intermediate region between the gray columns, more or less in the neighborhood of the nucleus dorsalis. The other degenerations in the anterolateral column in Figures r to w will be best understood by reference to the schematic section of the spinal cord, Figure 69. The degenerated fibers include the tectospinal and vestibulospinal tracts more or less intermingled, and a varying number from the anterior cerebrospinal tract. They include also homolateral cerebrospinal fibers. The positions of the degenerated tracts are best understood by imagining a blending of the descending degenerations seen in Figures 79, 77 and 83.

The descending degenerations in the posterior columns in Figures 81, r to w, all belong clearly to the same system; descending fibers from posterior nerve roots enter the spinal cord above the lesion, and their varying positions depend on the distances which they have traveled. Thus in Figure 81, r, the degenerated fibers representing the fasciculus interfascicularis are descending branches from nerve roots involved in the lesion or immediately above it. On the other hand the septal fibers of Hoche have come from some distance above the lesion, partly from the cervicobrachial region of the cord; our own sections from a case of destruction of the sixth and seventh cervical segments show this well. These descending fibers change their position rather curiously in a way to be explained only by the study of a complete set of serial sections. There is good reason for believing that they are all derived from the same source and that the fasciculus interfascicularis, Hoche's septal fibers, Hoche's bandalette, Philippe's sacral triangle, and Bruce's septomarginal bundle all contain descending root fibers only. Other methods show that the cornucommissural bundle (Fig. 81, t) is largely composed of association, propriospinal, fibers. (Compare Fig. 78, 1 to 3.)

Passing now to the degenerated tracts *above the lesion,* the student should refer again to the schematic section of the cord (Fig. 69). In the series of sections under consideration, Figures 81, p and o, above the lesion show degeneration in the posterior and anterior spinocerebellar tracts which are spread out along the margin on the anterolateral column. The anterior and posterior spinothalamic and the spino-olivary, spinotectal, and some shorter endogenous fibers which do not reach far above the level of the lesion make up the rest of the ascending degenerated fibers in the anterolateral column. None of these tracts except the posterior spinocerebellar are very well differentiated from their neighbors, but the posterior spinocerebellar fibers are marked by their definite position and large size, which shows well in the specimens but is not indicated in the figures.

In the posterior columns (Fig. p) the degenerations are at first very diffuse, perhaps partly due to local traumatism. In Figure o, about two or three segments above the lesion the degenerated areas are beginning to be limited to the fasciculi graciles and as far up as the cervical enlargement (Fig. n) the limitation to the fasciculus gracilis on either side is very marked. Ultimately these fibers are seen to end in the nucleus gracilis (Fig. k); a few end in the nucleus cuneatus.

The section through the middle of the inferior olive (Fig. j) is above the nucleus gracilis and nucleus cuneatus relaying the fibers of the posterior columns, so that the degeneration shows only in the upward continuation of the ascending fibers of the anterolateral columns. Some of these fibers apparently end in the inferior olives (spino-olivary fibers). The posterior spinocerebellar tracts join the restiform bodies (Fig. j). In Figure f the restiform body can be traced round the outside of the corpus dentatum and here it gives collaterals to the flocculus, to the cerebellar hemisphere, and to the corpus dentatum and nucleus fastigii. In Figure j, the group which mainly consists of the anterior spinocerebellar and spinothalamic fibers, and which is chiefly ventral to the spinal tract of the fifth nerve, appears to lose many fibers in the formatio reticularis of the medulla oblongata, perhaps in association with the nucleus lateralis and nucleus ambiguus. Other fibers of this group pass dorsally to the neighborhood of the fasciculus solitarius.

Higher up (Fig. e) this group of fibers is much diminished and forms a degenerated tract dorsal to the lateral margin of the medial lemniscus. It consists of fibers of the ventral spinocerebellar tract on their way to the vermis cerebelli by the roundabout road of the side of the brachium

conjunctivum and superior medullary velum (compare Figs. e, d, and 64). It also includes the dorsal and ventral spinothalamic fibers which end in the ventral nucleus of the thalamus and send collaterals to the substantia nigra (see Figs. c, b and a); lastly there are spinotectal fibers destined for both colliculi of the corpora quadrigemina (Figs. c and b). A few fibers from the endogenous neurons of the cord ascend in the medial longitudinal bundles (Figs. i to c) and probably form association bundles correlating trunk, limb, and eye muscles.

It is to be remembered that the great bulk of the ascending tracts from the cord to the thalamus are relayed in the nucleus gracilis and nucleus cuneatus and do not degenerate above these nuclei.

Late degenerations of the spinal cord are shown in Figure 82 from a patient who had a tumor of the dura completely interrupting the continuity of the fourth thoracic segment. The sections are stained by the Pal-Weigert method so that normal myelin shows dark staining and the myelin which has undergone degenerations takes no stain. A section through the tumor is shown in Figure 185. The Figure 82, d, marked third, fourth, or fifth thoracic, is in the immediate neighborhood of the lesion. It shows cavity formation and almost complete degeneration of all the constituents of the cord. The eighth thoracic segment shows degeneration of the oval field in the posterior columns. This tract consists of descending fibers from the posterior nerve roots. By the Marchi method these fibers can be traced all the way down the cord in suitable specimens (see Fig. 81, r to w), but they are usually too scattered to show by the Pal-Weigert method.

In the anterolateral columns degeneration is present in the lateral cerebrospinal, the rubrospinal, the vestibulospinal, and the tectospinal tracts; all these tracts have parent cells above the lesion. There is no degeneration in the anterior and posterior spinocerebellar, the anterior and posterior spinothalamic, the spinotectal, and spino-olivary tracts round the periphery of the cord, and the fasciculi proprii. All these tracts arise from cells below the lesion and therefore are not separated from their parent cells, so that they escape destruction.

In the twelfth thoracic segment there is no evidence of degeneration in the posterior columns, though in the early stages osmic acid stains would show scattered degenerated fibers. The anterolateral columns show degeneration of the lateral cerebrospinal, rubrospinal, tectospinal, and vestibulospinal tracts. The dorsal spinocerebellar tracts commence in the nucleus dorsalis cells of the first lumbar segment; they would not be degenerated and so they take a dark stain, yet they scarcely show in this

section through the twelfth thoracic segment, as they have just commenced and are still very small. Neither is there any evidence of the spinothalamic tracts as they do not concentrate into distinct tracts below the twelfth thoracic segment. At the level of the second lumbar segment all descending tracts except the lateral cerebrospinal have become too small or scattered to show by this method of staining.

Passing now to the degenerations above the tumor one finds that the cord just visible above the tumor is almost totally disorganized. There are only a few scattered myelinated fibers. The anterior gray column cells are in a condition of advanced chromatolysis. There is also a considerable amount of œdema as shown by many empty spaces, and there is a considerable increase in the number of neuroglia cells.

At the level of the first thoracic segment all tracts in the posterior columns with parent cells above the lesion are intact and show unaltered myelin. These are long and short fibers of the posterior roots arising from root ganglia above the lesion; they occupy the fasciculus cuneatus. On the surface of the fasciculus cuneatus some long fibers from thoracic root ganglia below the lesion have degenerated. In the anterolateral columns, the fasciculi proprii and ascending tract fibers from nerve cells above the lesion are not degenerated, and the lateral cerebrospinal, the rubrospinal, the tectospinal, and the vestibulospinal tracts, and fibers from the medial longitudinal fasciculus,—all descending tracts with parent cells above the lesion,—remain unaltered. In the posterior columns the fasciculi graciles, which consist of long fibers from posterior nerve roots of the sacral, lumbar, and lower thoracic nerves, show complete degeneration. Lying on the surface of the fasciculus cuneatus there are long degenerated fibers from the upper thoracic nerves below the lesion. The ascending tracts arranged round the periphery of the anterolateral column have disappeared. These are the spinocerebellar, the spinothalamic, the spinotectal, and the spino-olivary tracts and a few fibræ propriæ going from segments below the lesion to higher connections in the cord and brain stem. In these tracts nothing remains of those fibers which originate in cells below the lesion.

The condition of the third cervical segment is very similar. The degenerated long fibers of the fasciculus cuneatus, however, have been shifted from the surface to a position alongside of the fasciculus gracilis.

In the figures of the sections of the medulla oblongata one side only has been shaded; in the specimens the two sides are almost exactly alike.

At the lower end of the medulla oblongata the fibers of the fasciculus gracilis are completely destroyed, but here the degeneration ceases. Ulti-

mately atrophy of the nucleus gracilis and diminution of the fibers of the opposite medial lemniscus is to be expected, but this occurs slowly. There is no visible diminution of the long fibers in the funiculus cuneatus. There is marked loss of fibers in the dorsal and ventral spinocerebellar tracts. The other ascending tracts which arise below the lesion are too small and too much scattered at this level to show degeneration by this method of staining. The section through the restiform body (Fig. 82, i) shows a marked loss of fibers in its central part since it lacks these fibers of the posterior spinocerebellar tract which should join it from the lower thoracic segments.

In this material no degenerations were demonstrable in higher sections by this method of staining.

FŒTAL BRAIN STEM AND CORD

A great deal may be learned by examination of the cord and brain stem of fœtuses at various stages of myelination, as this shows in Pal-Weigert or iron hæmatoxylin specimens by difference in the intensity of the stain.

The tracts which are oldest phylogenetically myelinate earliest and stain most deeply. The nerve roots, the fasciculi proprii, the medial longitudinal bundle, the part of the medial lemniscus that belongs to the arm nerves, and the spinocerebellar tracts are all myelinated in a five and a half months' fœtus (Figs. 50, 51 and 52 from Alexander Bruce's Atlas). The part of the medial lemniscus belonging to the lower limb myelinates about two months later.

In a fœtus at term (Figs. 53 to 68), the nerve roots, the fasciculi proprii, the spinocerebellar tracts, the olivocerebellar tracts, the medial longitudinal bundles, the medial lemnisci and the lateral lemnisci (the lateral somewhat later than the medial), the restiform bodies, and the brachia conjunctiva are well or fully myelinated. The pyramidal tracts and their continuations are not myelinated until much later, and the corticopontine fibers and brachia pontis still later.

The fœtal sections may now be studied in greater detail. The cervical enlargement of a five and a half months' fœtus (Fig. 50) shows myelination of the anterior and posterior nerve roots, the fasciculi proprii, the fasciculus cuneatus (long and short fibers) and the fasciculus spinocerebellaris posterior. The fasciculus gracilis and the fasciculus spinocerebellaris anterior show very faint myelination. The cerebrospinal, the rubrospinal, the vestibulospinal, the tectospinal, and the spinothalamic tracts

are unmyelinated. In fact, only the tracts necessary for the more primitive reflexes are ready to functionate. In this relation it is significant that the mother commences to feel the movement of the fœtus in utero during the fifth month. Figures 51 and 52 are sections of the oblongata of the same specimen. They show that the ventral part of the medial lemniscus comes from the nucleus cuneatus. The fila of the hypoglossal and glossopharyngeovagus and accessory nerves are well myelinated.

Figure 53 from the *lumbar enlargement of a full-time fœtus* shows many groups of *anterior gray column cells* corresponding to the wide muscular distribution of the lumbar or upper sacral nerves. The anterior and posterior nerve roots, and fibers entering the gray matter from the short fibers of the posterior columns are easily seen. The *gelatinous substance* is abundant in correspondence with the wide cutaneous distribution of the sensory nerves of this region. The posterior white column is differentiated into a *superficial zone* of fibers which myelinate late and are destined to join the funiculus gracilis and end in the nucleus gracilis in the oblongata, and a *deeper area of short fibers* which myelinate earlier. These soon terminate in the gray matter of the cord, some a little higher than the level of entrance and some below it; each fiber bifurcates into an ascending and a descending branch. The differentiation of the superficial and deep zones is seen only in exceptional specimens which have been secured in the brief interval between the myelination of the short fibers and that of the long. The *posterolateral column* shows no trace of myelin except a few small bundles of ascending nerve roots of coarse myelination. In this connection it may be noted that where any of the posterior nerve roots are cut transversely, the bundles show many fibers which have not attained their myelin sheath at the time of birth. It is doubtful if a child at birth has any epicritic sense; it is probably insensitive to light, touch, heat and cold slightly above and below blood heat, and tactile discrimination. It must be remembered that in the adult a large proportion of the fibers of the posterior nerve roots and of the fibers in the posterolateral column are devoid of myelin. S. W. Ranson has shown that these fibers convey pain and believes that they convey also the more pronounced degrees of heat and cold, *i.e.*, protopathic sense. They do not stain by the Pal-Weigert nor by the iron hæmatoxylin method. At the level of this section the lateral cerebrospinal tract is small; like other descending tracts it has only the lower lumbar and sacral motor neurons to supply, and this and other tracts named in the drawing are but little advanced in myelination. The *anterolateral fasciculus proprius* is fully

myelinated and of large size to provide for the many spinal reflexes associated with the lumbar and sacral plexuses.

Figure 54 is well up in the thoracic region of the same fœtus, as is evident from the full development of the *posterior spinocerebellar tract.* It is drawn on the same scale as the other sections, magnified ten diameters from the original, but appears very small in comparison. This small size is due to the fact that the upper thoracic nerves supply only the intercostal muscles and a small segment of the spinal muscles and corresponding skin. Hence, there is at this level little gray matter, little gelatinous substance, and the fasciculi proprii and the short posterior nerve roots are small since they represent only a few muscles and a small skin area. The long fibers conveying muscle sense from the legs and abdominal muscles are well differentiated into a distinct *fasciculus gracilis* and the late myelination of this tract makes prominent the septal descending nerve roots and retrocommissural fibræ propriæ. The descending fibers from the posterior nerve roots form the septomarginal fasciculus, or "oval field." The retrocommissural fibers forming the cornucommissural bundle of advanced myelination are probably fibræ propriæ.

In the gray matter at the medial side of the posterior horn is the *nucleus dorsalis,* with many fibers from the funiculus cuneatus winding characteristically round it. The axons of the large cells of the nucleus dorsalis form the *dorsal spinocerebellar tract,* which is seen on the dorsolateral margin of the lateral white column in this section. Its fibers are large and myelinate early. *The intermediolateral cell column* forms a distinct lateral prominence in the gray matter. Its cells are small; its fibers leave by the anterior nerve roots to form the white efferent communicating rami of the sympathetic. They are present in the eighth cervical to the third lumbar segments, and a group for the pelvic viscera is found in the second and third, or third and fourth sacral segments. A few similar cell groups are found in the upper cervical region.

The *lateral cerebrospinal tract* is separated from the surface by the posterior spinocerebellar tract, and the other long ascending and descending tracts of the anterolateral column bulk more largely than in the section through the lumbar enlargement. The *funiculus gracilis* is differentiated from the *funiculus cuneatus* by its late myelination and its smaller fibers; it is formed by the shifting medialward of the long leg fibers.

The cervical enlargement (Fig. 55) of the same cord where it supplies the forearm and short muscles of the hand is *bulky* because of the great increase of gray matter corresponding to the many large muscles and wide

skin area represented and the corresponding heaping of the fasciculi proprii. In the cervical cord descending tracts are also large as they have to distribute fibers to all the lower parts of the body, and the ascending, long sensory tracts have been gathering bulk as they ascend. *The gelatinous substance* is abundant. There are many distinct groups of *anterior column cells.* No cells of the *nucleus dorsalis* nor of the *intermediolateral cell column* are present in this region.

The *funiculus gracilis* is rendered prominent by its delayed myelination and fine fibers and forms a bulging in the dorsum of the cord. The funiculus cuneatus shows a group of fibers of slightly delayed myelination and fine caliber on its dorsal surface. This consists of long fibers for muscle sense from the arm; it is destined to end by synapses round the cells of the nucleus cuneatus in the oblongata. The deeper short fibers of this column are fully myelinated; posterior nerve roots enter it to bifurcate and ascend and descend some distance before entering the gray matter. They are never seen to pass directly into the gray matter. From these fibers many terminal and collateral fibers enter the gray matter. The *posterior spinocerebellar tract* has reached its full size; in front of it is seen a tract of somewhat later myelination and finer fibers, the *anterior spinocerebellar tract.* Close by the anterior fissure is the *anterior cerebrospinal tract;* this myelinates late like the *lateral cerebrospinal tract,* which, as in Figure 54, is separated from the surface by the *posterior spinocerebellar tract.* The tract of early myelination along the posterior medial septum is the septomarginal fasciculus, or oval field. It consists of descending fibers of the sensory roots which entered some distance above the section on their way to lower segments of the cord. The other tracts seen in this section require no special mention.

The second cervical segment (Fig. 56) shows as its most prominent feature the *formatio reticularis of the cord;* this is composed of the crossed part of the pyramidal tract (compare Fig. 58 with Figs. 57 and 56) before it gets condensed and arranged into a distinct *lateral cerebrospinal tract.* Myelinated fibers from the opposite pyramid may be seen running into it. The gray network is composed of neuroglia, nonmyelinated fibers (unstained), and occasional nerve cells. The *funiculus gracilis* is well differentiated. The *funiculus cuneatus* shows a well-marked surface group of long fibers of fine caliber and late myelination; the deeper fibers, more advanced in myelination, consist mainly of short posterior root fibers from a few segments farther down. The *septomarginal fasciculus* is well marked. It is quite probable that these fibers in the upper cervical region are

largely derived from the sensory root of the fifth nerve, but not the spinal tract of this nerve, which is a tract of fine myelinated and unmyelinated fibers. The *gelatinous substance* is very abundant and on its surface the *posterolateral column* is supplemented by the *spinal tract of the fifth cranial nerve. Dorsal roots* of the second cervical nerve may be seen coursing through the gelatinous substance to the neck of the posterior gray column. On the right side is seen one of the emerging spinal roots of the *accessory (eleventh cranial) nerve.* Its roots arise from a group of cells which are placed rather dorsolaterally; they run first dorsally, then laterally, and emerge in series between the posterior roots of the cervical nerves and the denticulate ligament. The medial group of motor cells for the *second cervical nerve* probably supplies the muscles of the suboccipital triangle, and the more lateral group, the hyoid depressors. The well-marked decussating fibers at the bottom of the anterior fissure is the lower end of the pyramidal decussation. The other tracts require no special comment.

The section through the *lower end of the pyramidal decussation* (Fig. 57) demonstrates the relation of the decussation to the peculiar bundling of the lateral cerebrospinal tract which contributes to the formation of the upper end of the formatio reticularis of the cord. The *posterior spinocerebellar tract* is concentrated just in front of the *spinal tract of the fifth nerve* and the gelatinous substance; more ventrally the *anterior spinocerebellar tract* is well differentiated; it myelinates a little later and is finer in fiber. In the section from which Figure 54 was drawn this tract has probably been a little too much bleached. The *funiculus gracilis* shows the commencing *nucleus gracilis* (Fig. 57).

A section through the *middle of the pyramidal decussation* is shown in Figure 58. Here three-fourths, more or less, of the pyramidal tract crosses to form the *lateral cerebrospinal tract* which may be seen to commence as the longitudinal fibers of the reticular formation. In crossing, the pyramidal fibers cut off the anterior part of the anterior gray column. The ventral cells in this detached part of the anterior gray column give rise to the *first cervical nerve.* The dorsal cells give rise to one of the roots of the *accessory nerve.* The *gelatinous* substance is very abundant; a large area on its surface is occupied by the *spinal tract of the fifth nerve,* which is marked by late myelination and the fineness of its fibers. The position, the fine fibers and the late myelination of this sensory tract is strikingly similar to the dorsolateral fasciculus with which it appears to be in series (see Figs. 53, 54 and 55). The *nucleus gracilis* begins to

show *internal or deep arched fibers,* leaving its ventral surface; the *nucleus cuneatus* is beginning to appear. The *fasciculus spinocerebellaris posterior* is a concentrated bundle triangular on section. The other tracts do not differ much from their appearance in previous sections.

In the section through the closed part of the medulla oblongata at the *lower end of the inferior olive* (Fig. 59) the most noticeable features are the great development of the *gracile and cuneate nuclei* and the *deep arcuate fibers* streaming from their ventral surface to intercross and form the opposite *medial lemniscus.*

On the right side of the specimen the *funiculus gracilis* has almost completely ended by arborizing round the cells of the *nucleus gracilis.* The *funiculus cuneatus* has still many fibers left. From the cells of the gracile and cuneate nuclei the second relay of fibers for these tracts of muscle sense sweep round ventrally and medially. They are the *deep arcuate fibers.* Intercrossing with their fellows of the opposite side, they form a very definite tract of advanced myelination which lies medial to the inferior olive and dorsal to the pyramid; this tract is the *medial lemniscus,* which ascends to the ventrolateral nucleus of the optic thalamus. The fact that these deep arcuate fibers form the opposite medial lemniscus receives striking confirmation in this way. In many cases of syringomyelia these fibers are cut across by the gliomatous growth and by cavitation as neatly as if cut experimentally by a knife. When this occurs the opposite medial lemniscus disappears in proportion to the interruption of these deep arcuate fibers. In the oblongata the inferior olives confine these tracts between them and give them the characteristic ribbon-like shape on section.

The opposite lemniscus is not the only upward relay of the funiculus cuneatus. Among its fibers dorsal to the nucleus cuneatus is an *accessory cuneate nucleus* (Fig. 59, left side); in this many fibers of the funiculus cuneatus end. From the dorsal surface of this accessory nucleus fibers stream lateralward to join the restiform body and thus connect this tract with the vermis cerebelli of the same side. The left side of the drawing (and the section from which it was drawn) seems to show a similar connection of the funiculus gracilis, but this is denied by many. According to André Thomas, these *posterior superficial arcuate fibers* establish with the cerebellum a connection for the arm and neck muscles similar to that established for the leg muscles by the anterior and posterior spinocerebellar tracts.

The *spinal tract of the fifth nerve* and the corresponding abundance

of the *gelatinous substance* is a prominent feature of the part of the section ventral to the nucleus cuneatus. It is this massing of gelatinous substance that forms the *tuberculum cinereum* on the surface of the oblongata (Fig. 11). Leaving the medial surface of the gelatinous substance are fibers which form a second relay for the sensory root of the trigeminal nerve. They cross the median line and form a tract lateral to the medial lemniscus and dorsal to the olive, called the *trigeminothalamic tract* (compare with Fig. 15, where they are represented diagrammatically). Ventral to the gelatinous substance the *posterior spinocerebellar tract* forms a well-marked tract of coarse fibers and advanced myelination; from this fibers stream dorsally as it is shifted backward to join the restiform body. The *anterior spinocerebellar tract* is medial to this and a little anterior; its fibers are somewhat scattered. The *rubrospinal tract* is ventromedial to the gelatinous substance (Fig. 15). Its fibers are rather fine and are in about the same stage of myelination as the pyramids. Immediately dorsal to the lemniscus lies the *tectospinal tract.* It is a tract of fine fibers and late myelination and in this particular fœtal cord shows hardly any myelination. It shows well in Figures 59 and 60 and in Figure 15. Dorsal to this, and just ventral to the central canal is a tract of coarse fibers and advanced myelination (compare with Fig. 15). It is the *medial longitudinal fasciculus,* and will be fully described in connection with Figure 60.

The *pyramids* are two compact bundles of late myelination lying ventral to the medial lemniscus and between the olives and the anterior median fissure (compare Fig. 15). It is not possible to demonstrate anatomically that they contribute fibers to the opposite nucleus of the twelfth nerve, but physiological and pathological phenomena show that either directly or indirectly such a connection is established. There is a lesser connection with the twelfth nucleus of the same side. The *anterior and posterior spinothalamic tracts* ascend through the reticular formation dorsal to the olives; they myelinate late (compare Fig. 15). On the ventral surface of the pyramids are seen the *anterior superficial arcuate fibers* and their nuclei. They belong to the system of tranverse fibers and nuclei of the basilar portion of the pons.

The *inferior olives* will be described in connection with Figure 60. In this figure cells are indicated diagrammatically by dots in the gray matter of the olives. They show well in iron hæmatoxylin specimens.

In the oblongata there are many groups of nerve cells whose connections are not known; here and in higher sections only those will be

described whose definite anatomical connections and physiological functions are known and give them clinical significance.

By the side of the central canal is the prominent *nucleus of the twelfth or hypoglossal nerve,* the motor nerve to the tongue. Issuing from its ventral surface are the fila of this nerve. Winding around the cells are fine medullated fibers, probably association fibers from such nuclei as the sensory nuclei of the fifth, ninth and tenth cranial nerves. The hypoglossal nerve passes ventrally through the olive and side of the pyramid to emerge between these structures (compare Fig. 14).

Dorsolaterally to the twelfth nucleus is the small-celled *dorsal nucleus of the ninth and tenth (glossopharyngeal and vagus) nerves;* lateral to this nucleus is a rounded bundle of longitudinally directed fibers called the *tractus solitarius.* This bundle, prominent in all sections of the lower oblongata (compare Figs. 14 and 15) is the spinal tract of the glossopharyngeal nerve. The nucleus associated with it is the *gustatory nucleus;* in it end the gustatory fibers of the glossopharyngeal and chorda tympani nerves. From its neighborhood issue fibers of the glossopharyngeal nerve to emerge lateral to the olive (compare Figs. 14 and 15); from the medial side arcuate fibers go to the opposite lemniscus medalis or more likely to a neighboring region in the formatio reticularis. The rather large cells of the *nucleus ambiguus* form a small group medial to the gelatinous substance and close to the emerging and entering roots of the tenth nerve. This is the somatic motor nucleus of the glossopharyngeal, vagus, and accessory nerves (compare Fig. 14).

The *formatio reticularis* remains to be described. In well stained sections of the oblongata the area which lies between the raphe medially, the inferior olive ventrally, the central gray matter dorsally, and the great nuclear masses and nerve tracts laterally shows under the microscope a complex interlacing network or reticular formation. In Pal-Weigert and iron-hæmatoxylin specimens the longitudinal nerve fibers show as dark dots (circles under higher powers) in some places massed together, in others more or less equally distributed, varying much in size, and in fœtal specimens varying also in the degree of myelination. The transverse fibers arch gracefully and most of them have been described under the name of the *internal arcuate fibers,* but others less pronounced join the sensory nuclei of the cranial nerves with the medial lemniscus, the medial longitudinal fasciculus, or the trigeminothalamic fasciculus. Amongst the transverse fibers, too, must be mentioned fibers going from the pyramidal tracts to the opposite motor nuclei of cranial nerves. These fibers are

few and scattered, and are taken for granted on physiological grounds, not demonstrated anatomically. Of the longitudinal fibers the medial longitudinal fasciculus, the fasciculus tectospinalis and the medial lemniscus have been noted. Mention must also be made of the fasciculus rubrospinalis and the vestibulospinal, spinothalamic, spinotectal, and anterior spinocerebellar tracts. These last all myelinate late and the only one which can be differentiated in the fœtal specimens is the *anterior spinocerebellar tract.* Their positions have been indicated in Figure F of the long diagram. In the lateral part of the formatio reticularis many nerve cells are found scattered and in groups. Hence the lateral part is spoken of as the gray reticulation (*reticularis grisea*) to distinguish it from the medial portion which is without nerve cells and is called the *reticularis alba.* A similar reticular formation is found in the tegmental portions of the pons and mesencephalon.

The section drawn in Figure 60 is fortunate in the way it shows the *restiform body,* and the *olivocerebellar connections,* the *roof nuclei* of the cerebellum and the *nuclei and connections of the vestibular nerve.* It is very much the same as Figure 15, with which it should be compared, but passes more obliquely backward and upward from the middle of the olive ventrally, to the fissura prima of the superior vermis cerebelli dorsally. The *pyramid,* the *medial lemniscus,* the *anterior spinocerebellar and rubrospinal tracts* need no special description in this figure. The *tectospinal* tract is marked by the contrast of its fine fibers with the coarse fibers of the surrounding tracts. The *medial longitudinal fasciculus* is rendered very prominent in all fœtal brain stems by its early myelination and by contrast with the fine and scattered tectospinal tract in front of it. It can be traced from the upper end of the mesencephalon (Fig. 68) to the upper end of the pyramidal decussation where it joins the anterior fasciculus proprius. It is largely formed from the cells of Deiters' nucleus, but this can be demonstrated only by degeneration methods as already described.

The inferior olive forms a very prominent feature of this section. In Pal-Weigert specimens which are not counterstained it shows as it is drawn here with fibers only, but in counterstained specimens, and in those stained by the iron hæmatoxylin method, very numerous, rather large cells show its importance as a nucleus. In Ramon-y-Cajal's drawings and in Golgi silver specimens, the cell dendrites and the network of fibrils forming synapses with them are of marvelous complexity. (See Ramon-y-Cajal or Quain.) Comparison of the olive in section (Fig. 60) with its recon-

struction in Cunningham's Textbook shows that it is a sheet of gray matter folded up in the oblongata, with the hilum toward the median plane. Two separate sheets lying medial and dorsal to the main olive are called *medial and dorsal accessory olives* (Fig. 14). *Olivocerebellar fibers* flow out of the hilum of the right olive (Fig. 60) decussate in the median line with their fellows of the opposite side, and stream through the left olive to reach the left restiform body where they form a great mass of fibers. These myelinate later than the spinocerebellar and cuneocerebellar fibers which they surround; they are well seen in Figure 60, with which should be compared Figure 14. Most of them run ventral to the gelatinous substance, but many fibers higher up pass through the spinal tract of the fifth nerve. Many longitudinal fibers show in cross section on the surface of the olives. Some of those on the dorsal surface of the olive come from the spinal cord, forming the *spino-olivary tract.* The rest are of undetermined origin; probably they come from the *central tegmental tract.* When one cerebellar hemisphere is absent or atrophies, the opposite inferior olive atrophies with it (Fig. 84), but though the inferior olives are so prominent anatomically and are evidently intermediate stations between the cerebrum and cord and the opposite cerebellar hemisphere, their function is entirely unknown. Batten and Holmes (*Brain,* Vol. 35, page 269) have shown that many endogenous fibers springing from cells of the spinal cord end by arborizing round cells of the inferior olive and probably of the dorsal accessory olive (Fig. 78, 7). Thus the inferior olives may form a relay station between the spinal cord and the opposite cerebellar hemisphere.

The restiform body, seen in Figure 60, consists of two constituents which myelinate at different times. The central group of fibers myelinating early is composed of the *posterior spinocerebellar tract* and *posterior superficial arcuate fibers* from the accessory cuneate nucleus, both of the same side. This element sweeps round the lateral surface of the dentate nucleus to the superior vermis where some of the fibers decussate and all end. Some fibers or collaterals go to the flocculus and corpus dentatum. The peripheral fibers in the restiform body myelinate later. They are the *olivocerebellar fibers* and pass to the lateral hemisphere of the cerebellum.

Medio-ventral to the restiform body the *spinal tract of the fifth nerve* is prominent; its nucleus, the upper end of the gelatinous substance, lies medial to it. Medial to this and well shown in this section is the *rubrospinal tract* at about the same stage of myelination as the pyramidal tract.

Passing dorsalward between the restiform body and the spinal tract of the fifth nerve is the *vestibular nerve.* It is seen passing to nuclei in the lateral part of the floor of the fourth ventricle. This is the sensory nerve from the semicircular canals, the saccule and the utricle of the ear, which together form the *organ of equilibration.* Its fibers are axons of the vestibular ganglion situated in the internal auditory meatus. Entering the oblongata, they end in dorsal and lateral nuclei. The fibers split into short ascending and longer descending branches. The ascending fibers end in an upward prolongation of the lateral nucleus. The descending fibers form a well-marked descending tract with a nucleus of cells scattered among them. Of the two chief nuclei the *lateral or Deiters' nucleus* is of great clinical importance; from its lateral side fibers pass to the vermis cerebelli. Fibers from the *roof nuclei* of the cerebellum also connect with Deiters' nucleus; indeed the roof nuclei, especially the *nuclus fastigii,* are probably an essential part of the vestibular nervous mechanism. Besides taking part in the function of equilibration Deiters' nucleus appears to have a most important function in stimulating muscle tone. The vestibular nuclei are the first part of the cerebellum to appear in the vertebrates; with the roof nuclei they form pretty nearly the whole cerebellum in amphibia, and children may reach maturity without any part of the cerebellum developing except these vestibular centers. From the medial side of Deiters' nucleus fibers are seen (Fig. 60) crossing medially to join the *medial longitudinal bundles* of both sides, while others form the *vestibulospinal tract* of the same side (compare Fig. 77, 1). This tract myelinates later than the medial longitudinal bundle and consists of finer fibers. Its position in the cord has been described. At the level of the section under consideration it lies medial to the rubrospinal tract. The vestibular fibers of the medial longitudinal bundle and the vestibulospinal tract reflexly coördinate eye, head, and body movements with equilibrating stimuli from the vestibules and semicircular canals; they also maintain muscle tone. Through the connection between the nucleus fastigii and Deiters' nucleus the vestibulospinal tract may also form one of the efferent tracts from the cerebellum to the spinal cord. Normally they are under the control of the motor cerebral cortex and globus pallidus (palæostriatum) through the cerebrospinal and pallido-rubrospinal tracts. If this control is removed they are partly responsible for the muscular rigidity in the various forms of spastic paralysis.

By the side of the fourth ventricle a small portion of the *dentate nucleus* of the cerebellum is seen. Along the medial side of the nucleus

are the commencing bundles of the *brachium conjunctivum* which is formed by the axons of its cells. Ventrolateral to the restiform body is the *cochlear nerve* and the *ventral cochlear nucleus* (compare Fig. 61).

In the roof of the fourth ventricle lie the *roof nuclei* (also called the nuclei fastigii or nuclei tecti) of the cerebellum. Lateral to the nucleus fastigii is the *nucleus globosus,* and still more lateral, the *nucleus emboliformis,* which may be regarded as a detached part of the corpus dentatum. The *nucleus globosus* and *nucleus fastigii* receive fibers from the vermis and send cerebellar efferent fibers which pass by the side of the vestibulospinal tract to the vestibular nuclei and to the spinal motor neurons (André Thomas).

The section through the *middle of the pons,* shown in Figure 63, is cut nearly parallel with the upper border of the pons and passes through the root of the fifth nerve. It should be compared with the adult section (Fig. 61), which is somewhat lower down and more vertical to the axis of the brain stem, running in the direction of the brachium pontis. The section in Figure 63 runs obliquely through the upper part of the brachium.

Even to the naked eye the pons on section is divisible into two parts, a dorsal *tegmental portion* and a ventral *basilar portion;* the two are separated at the lower end by the deep transverse fibers of the *corpus trapezoideum* (Fig. 62).

The *basilar portion* consists of longitudinal and transverse fibers; scattered among the transverse fibers are an enormous number of nerve cells not shown in the figure; these cells together form the *nuclei pontis.* In the figure only the longitudinal fibers which form the pyramidal tracts are seen well as they alone are myelinated at birth; the transverse fibers are faintly indicated. The *pyramidal fibers* are gathered into a compact bundle on each side in preparation for their entry into the oblongata. On physiological evidence it is thought that they here give off fibers to the opposite facial nucleus for the supply of all the facial muscles and that they also give fibers to those cells of the facial nucleus on the same side which supply the orbicularis oculi and frontalis; this means that the lower facial muscles have only a contralateral pyramidal supply, but the upper facial muscles have a double pyramidal supply, both ipsilateral and contralateral. Fibers are also given off to the motor nuclei of the fifth nerve of the same as well as the opposite side. The pyramidal tract is believed to supply fibers to the homolateral, as well as the heterolateral, fifth and upper seventh motor nuclei because when the face and trunk are paralyzed on the right side, for example, by a hemorrhage into the

left internal capsule, the muscles of mastication on the right side escape and both eyes can be closed together, though the right eye cannot be closed separately. The *sixth nucleus* is supplied with pyramidal fibers from the opposite pyramidal tract only.

The *transverse fibers* of the basilar portion are seen assembling to form the *brachium pontis*. Comparison of Figure 68 with Figures 22 and 23 will show that the medial and lateral parts of the basis pedunculi are not myelinated in the full-time fœtus. The medial fifth of the basis pedunculi consists of fronto-pontine fibers from the frontal cortex, and the lateral fifth of temporo-pontine fibers from the temporal cortex; these myelinate late. They form two-fifths of the longitudinal fibers entering the upper end of the pons from the mid-brain. They arborize round the cells of the nuclei pontis and these cells send a fresh relay of fibers to the opposite lateral hemisphere of the cerebellum by way of the *brachium pontis*. Thus each hemisphere of the cerebrum is intimately connected with the opposite hemisphere of the cerebellum by fibers relayed in the nuclei pontis. The cells of the nuclei pontis are not shown in the figure; they do not show in Pal-Weigert stains unless counterstained, but they show well in iron hæmatoxylin stains and in counterstained sections.

Corpus trapezoideum. Between the pars basilaris pontis and the pars tegmentalis pontis at the lower end of the pons there is a group of deep transverse fibers which are fully myelinated at birth, and hence form a prominent object in the fœtal brain stem. This is the corpus trapezoideum (compare with Figs. 61 and 62). Reference to Figure 102 will help to explain it. It forms the second relay of fibers for the auditory nerve, comes from the ventral and dorsal cochlear nuclei, mainly from the ventral nucleus, and passes to the other side of the pons, where it turns upward and becomes the *lateral lemniscus* (see Fig. 64). The lateral lemniscus partly ends in or sends collaterals to a nucleus embedded in it called the *superior olive* (Fig. 63; see also Fig. 62). In the section under consideration the *superior olive* is seen to send fibers dorsalward to the nucleus of the sixth nerve and probably to the medial longitudinal bundle; thus there is formed a reflex route by which the eyes and head can be quickly turned toward the side from which an auditory impression is received by the cochlear nerve. By means of this special short reflex path an animal hearing a sound, friendly or otherwise, immediately turns its head and eyes toward the side whence the sound comes; the value of such a reflex is obvious.

At the lower end of the pons the *medial lemniscus* adapts itself to the

new arrangement of fibers and becomes a prismatic bundle ascending through the medial part of the corpus trapezoideum (Figs. 63 and 62).

On either side of the middle line, close to the floor of the fourth ventricle, the *medial longitudinal fasciculus* forms a band well marked in the section because of its early myelination. It receives fibers from the *nucleus of the sixth nerve* close beside it; these go by way of the medial longitudinal bundle to the fourth and third nuclei and thus provide for conjugate movements of the eyes by intimate interconnection of the third, fourth, and sixth nuclei. The medial longitudinal fasciculus is continued downward into the cervical region of the cord as part of the anterior fasciculus proprius and provides for the turning of the head in correspondence with eye rotatory movements. It effects this through connections with the eleventh cranial and the upper cervical nerves.

The rest of the tegmentum pontis presents a well-marked *reticular formation;* among the longitudinal fibers are found the *rubrospinal, tectospinal, spinothalamic* and *trigeminothalamic fasciculi* (compare with Fig. H of the long diagram). The position of the *anterior spinocerebellar tract* is medial to the gelatinous substance and among the fibers of the corpus trapezoideum. Many of the transverse fibers are passing from the sensory nuclei of the fifth nerve to the opposite side where those from the lateral sensory nucleus join the medial lemniscus and those from the gelatinous substance join the *trigeminothalamic tract.* The sensory nuclei of the fifth nerve also send association fibers to the third, fourth, sixth, seventh, and twelfth nuclei (motor nuclei to the eye, face and tongue) and perhaps to motor nuclei of the other cranial nerves and those spinal nerves at least which control head movements. These association fibers provide for many facial and eye reflexes.

The *brachium conjunctivum,* which in the previous section was forming by the medial side of the nucleus dentatus of the cerebellum, in this section is a prominent band of early myelination which lies by the side of the fourth ventricle and helps to form its lateral boundary. It consists mainly of the axons of the cells of the nucleus dentatus. This nucleus is an intermediate station between the cortex of the cerebellar hemispheres and the red nucleus and thalamus of the opposite side and to these the brachium conjunctivum may be traced.

The *root of the fifth cranial nerve* is seen in this section as it passes through the brachium pontis or middle cerebellar peduncle. The fifth nerve is motor to the muscles of mastication and sensory for the anterior part of the scalp, the face and eye, and the mucosa of the nose, palate and

anterior two-thirds of the tongue. It conveys impressions of touch, heat and cold, pain and muscle sense for this area. Its nuclei will be more easily understood if they are correlated with those which enter into the formation of a typical spinal nerve. For example, a typical nerve in the cervical region (see Figs. 55 and 69) has, first, a *motor root* starting as the axons of motor cells in the anterior gray column; second, a *sensory root* starting as the axons of a spinal ganglion. These sensory axons enter the cord and bifurcate. Besides sending many collaterals to form relations with neighboring motor and sensory cells, some fibers of the sensory root end by arborizing round the cells of the gelatinous substance and perhaps other cells of the posterior horns. By a new crossed relay these cells convey impressions of touch, heat, cold, and pain. Other fibers of the sensory root (carrying muscle sense, etc.) ascend to the nucleus cuneatus and are thence relayed to the opposite optic thalamus by the medial lemniscus. The accessory cuneate nucleus also establishes homolateral cerebellar connections for the upper sensory nerves.

Nuclei of the fifth nerve. In this section (Fig. 63) connections corresponding to all these are established for the fifth nerve. On the medial side of the root near the floor of the ventricle is a group of large multipolar cells which is the *masticatory (motor) nucleus.* Its axons form the *motor root.* From both pyramidal tracts, but chiefly that of the opposite side, it receives fibers which synapse with its cells directly or through intercalated cells. It also receives collaterals from both sensory nuclei of the fifth nerves, and from nuclei of other cranial nerves. Between the root of the fifth nerve and the corpus trapezoideum is seen the upper end of the *spinal tract of the fifth nerve* and the gelatinous substance which forms one of its sensory nuclei. This has been seen extending down into the upper cervical cord. Fibers for heat, cold, and pain, and perhaps epicritic touch end in this nucleus and are relayed by arcuate fibers to the opposite *trigeminothalamic tract* (see Fig. 15). Dorsolateral to the entering root is the *lateral sensory nucleus.* This is so like the nucleus cuneatus in its structure that the writer regards it as the end nucleus for those fibers of the fifth nerve which convey muscle, tendon, and joint sense, probably not only for the muscles of mastication, but also for the muscles of the face and tongue.

Second sensory neurons of the fifth nerve. From the lateral side of the lateral nucleus fibers pass to the vermis cerebelli. From its medial side arcuate fibers for muscle sense pass to the dorsal surface of the oppo-

site *medial lemniscus.* The *trigeminothalamic tract,* formed by axons from the nucleus of the opposite spinal tract of the fifth nerve, is the relay to the thalamus for heat, cold, and pain. It is distinct from the posterior spinothalamic tract and lies nearer the middle of the formation reticularis. This is proved by certain limited pontine lesions, as from embolism or thrombosis of the posterior inferior cerebellar artery, where one of these tracts may escape while the other is involved. The trigeminal sensory nuclei are freely connected by collaterals with the motor nuclei for all the muscles of the areas covered by the sensory supply (seventh, ninth, and twelfth cranial nerves), and perhaps also with the third, fourth, and sixth cranial nerve nuclei and at least those nuclei of spinal nerves which more immediately control head movements.

The *mesencephalic root of the fifth nerve.* In the section (Fig. 63) one root is seen passing dorsalward by the medial side of the brachium conjunctivum. It is the *mesencephalic root,* but as nothing is known of its function, it will not be described further.

The *sixth or abducent nucleus and nerve.* The emerging fibers of the sixth nerve, motor to the rectus oculi lateralis which turns the eye outward, pass ventrally through the pons a short distance from the median plane. This root is seen to arise from the medial side of a group of large multipolar nerve cells a short distance ventrolateral from the medial longitudinal bundle. This cell mass forms the nucleus of the sixth nerve; it lies under the eminentia medialis (Fig. 11); this has been improperly called the colliculus facialis, but the prominence is produced by the sixth nucleus and not by the seventh root. (See also Figs. 12 and 24). The connection between the superior olive and the sixth nucleus here called the *peduncle of the superior olive* has already been described and its significance indicated in describing the corpus trapezoideum (see Fig. 24).

Mechanism for conjugate deviation of the eyes. Association neurons in the sixth nucleus send axons into the medial longitudinal fasciculus, which ascend to the third nucleus in the mesencephalon (Fig. 68). The third nucleus sends some axons across to the opposite third nerve; these probably supply the medial rectus muscle for the purpose of conjugate deviation. A lesion of the right sixth nucleus not only causes paralysis of the right external rectus, but the left internal rectus is also paralyzed for conjugate deviation to the right; a lesion of the right medial longitudinal bundle, when the right sixth nucleus is intact, results in inability to turn the left eye inward in the movement of conjugate deviation to the right.

The sixth nucleus is controlled by the opposite pyramidal tract, and irritation of the posterior end of the middle frontal convolution causes conjugate deviation of both eyes to the opposite side.

Nucleus of the seventh or facial nerve (Fig. 60). Medial to the spinal root of the fifth nerve (compare Figs. 62 and 24), and dorsolateral to the superior olive is a large-celled nucleus which is the motor nucleus of the seventh nerve, the motor nerve to the muscles of the face. It is in series with the nucleus ambiguus seen in Figures 12, 24 and 59. Streaming from the dorsal surface of this nucleus are many rather scattered fibers which form the fila of the seventh nerve. They take a curious course, passing dorsomedially, lateral to the sixth nucleus (see Fig. 24) to reach the floor of the fourth ventricle and thence medially over the dorsum of the sixth nucleus. In sections at other levels the fila of the seventh nerve may pass medially to the sixth nucleus. The fibers then unite to form a compact bundle which runs cephalward as a rounded bundle dorsolateral to the medial longitudinal bundle (see Figs. 24 and 62). Soon this again turns lateralward and then in many bundles passes ventrolaterally medial to the spinal tract of the fifth nerve and the gelatinous substance and lateral to the upper end of its own nucleus, and slanting caudalward, finally emerges between the oblongata and pons (Figs. 61, 62).

The *splanchnic efferent nucleus of the seventh nerve* (Figs. 62 and 63). In some sections at this level one may find a small rounded group of smaller cells dorsal to the large-celled nucleus. This is the splanchnic efferent nucleus of the seventh nerve and supplies secretory fibers to the salivary and lachrymal glands.

Connections of the seventh nucleus. The large-celled somatic nucleus receives collaterals from the fifth nerve and its nuclei and cortical fibers from the pyramidal tract of the opposite side. That part of its nucleus which acts on the muscles of the brow and eyelid also receives homolateral pyramidal fibers.

Sensory root of the seventh nerve. The seventh nerve has a sensory root called the *nervus intermedius* which can only be studied by special methods. Its peripheral fibers form the afferent part of the chorda tympani and carry the sense of taste for the anterior two-thirds of the tongue. The nervus intermedius also has a variable number of fibers for ordinary sensation to parts of the concha and auricle and sometimes the soft palate and tongue. Its cells are in the geniculate ganglion and its central portion ends in the *fasciculus solitarius for taste,* and in the *spinal tract of the fifth nerve for pain, heat, and cold.*

This is an exceedingly important section when it comes to the explanation and diagnosis of lesions of the pons, and this figure (60) and Figures 12, 24, 61, 62 and 63 should be studied till all the structures described and their interrelations become very familiar.

The section shown in *Figure 64* passes obliquely through the *upper end of the pons and brachia conjunctiva* with the *anterior medullary velum* just where the *fourth nerves decussate* (compare Figs. 17 and 18). On the left side, which is at rather a lower level than the right, the upper edge of the root of the *fifth nerve* has been caught as it enters the brachium pontis. In the basilar portion of the pons the *pyramidal tract* is seen, broken up into many fasciculi by the transverse pontine fibers. Between the basilar and tegmental regions the arrangement of the fibers of the *medial lemniscus* has changed again so that it assumes in transverse sections the appearance of a horizontal band. Lateral to this, the *lateral lemniscus,* a tract of finer fibers and later myelination, now takes distinct form. In the preceding section (Fig. 63) this tract can barely be distinguished between the superior olive and the gelatinous substance, as the fibers of the corpus trapezoideum assume a cephalward direction at its lateral end. A little higher (Fig. 64) it is a distinct tract which sweeps obliquely backward on the surface of the brachium conjunctivum on its way to the inferior colliculus (Figs. 65 and 66) and to the medial geniculate body (Fig. 68). In Figure 64 the cells of its nucleus are seen among its fibers. In man and the orang, the nucleus has been shown to be continuous with the superior olive; it is probably a relay station for auditory reflexes.

Aberrant pyramidal fibers. At this level and a short distance above and below it small rounded bundles of fibers finer than those of the medial lemniscus can be seen embedded in its ventral surface near its medial border. They are in the same stage of myelination as the pyramidal fibers, and are in fact pyramidal fibers destined for the motor nuclei of cranial nerves. Some of them may reach the cord for the nuclei of the eleventh cranial nerve and the first and second cervical nerves, which supply head-turning muscles. They go mainly to crossed motor nuclei, but partly to homolateral nuclei of the seventh, eleventh, and twelfth nerves. Perhaps the best name for these fibers is that used by Dejerine—the *aberrant pyramidal fibers.*

The *brachium conjunctivum* is more deeply placed in this than in the last section; it bounds the formatio reticularis laterally. Dorsal to the brachium conjunctivum on the right side is seen the *anterior spino-*

cerebellar fasciculus. It descends from here in the superior medullary velum to end in the vermis.

The *fourth or trochlear nerve.* The *decussation of the fourth nerve* in the anterior (superior) medullary velum, and its descending portion are seen in this section (Fig. 64). Part of its nucleus is seen in Figure 66 (compare Figs. 12, 23, 22 and 18 in this order for nucleus, mode of origin, descending portion, decussation, and emergence of the nerve). The *trochlear nerve* is the motor nerve to the superior oblique muscle of the eye. It arises from a cylindrical group of large multipolar cells, embedded in the dorsal surface of the medial longitudinal fasciculus beneath the upper end of the inferior colliculus. The nerve fibers spring from its lateral surface (Figs. 66 and 22), pass dorsolaterally round the aqueductus cerebri, run caudad (Figs. 64 and 18) to reach the lower end of the inferior colliculi and then decussate and emerge from the dorsal surface of the superior medullary velum (Figs. 64 and 18). The apparently complete decussation and the peculiar course are not comparable with the course of any other cerebral or spinal nerve. The intercrossing of the fourth nerves suggests that their nuclei must be controlled by the homolateral pyramidal tract, or they may be controlled entirely through the medial longitudinal bundle from the nucleus of the third nerve. This is the more likely as there is no separate voluntary control of the oblique muscles, and their use is to correct the oblique action of the superior and inferior recti.

At this level the *tectospinal tract* lies in the formatio reticularis just ventrolateral to the medial longitudinal fasciculus; the *rubrospinal tract* lies dorsal to the medial end of the medial lemniscus (see Fig. K, long diagram). In the *medial longitudinal fasciculus* fibers from the vestibular and the sixth nuclei ascend to form connections with the fourth nuclei; and at a higher level with the third. In the tegmentum, dorsal to the lateral end of the medial lemniscus, lie the *trigeminothalamic and spinothalamic* tracts. These tracts carry pain, heat, and cold, and perhaps touch for the whole of the opposite side of the head, face, and body. The *medial lemniscus* conveys muscle, tendon, and joint sense,—sense of active motion and passive position, and bone or tuning-fork sense, from the opposite side of the body. A little higher these three tracts join, and at this higher level a complete lesion of the medial lemniscus causes loss of all these forms of sensation for the opposite side of the whole body. The *lateral lemniscus* represents hearing; it is not entirely composed of crossed connections, so that complete deafness of one ear must be due to a lesion of the ear itself or of the auditory nerve before it is relayed by

the corpus trapezoideum or if at this level to lesions of both lateral lemnisci. The cerebral tract for the sense of equilibrium from the vestibular nucleus is believed to end in the middle and inferior temporal gyri. The position of the tract as it ascends is not known.

Figure 65 passes obliquely through the upper end of the pons and the inferior colliculi and is introduced to show the ending of many of the fibers of the *lateral lemniscus* in the inferior colliculi. It is the function of the inferior colliculi to coördinate eye and body movements with impressions of hearing. The afferent tract for this purpose is provided by the collaterals of the lateral lemniscus which end by forming synapses with the dendrites of the cells of the inferior colliculus. The efferent tract from the cells of the inferior colliculus may be by way of the tectospinal tract, but this path is not established. The commencement of the tectospinal tract in the superior colliculus will be seen in Figure 68. The *brachia conjunctiva* are here commencing to decussate, the *medial lemnisci* are shifting lateralward. The *fourth ventricle* is narrowing as it joins the aqueductus cerebri.

Figure 66 is also an oblique section a little higher up than Figure 65, in fact through the *upper end of the inferior colliculi;* it shows the *decussation of the brachia conjunctiva* (compare with Fig. 22). Longitudinal fibers of the same stage of myelination may be seen passing between the decussating bundles; these are fibers that have crossed and are again taking a longitudinal direction (Fig. 22).

The *medial lemniscus* is shifted still more lateralward on its way to the thalamus past the side of the red nucleus. Bundles of *aberrant pyramidal fibers* may be found embedded ventrally in the medial border of each medial lemniscus. The *lateral lemniscus* appears to have contributed much of its bulk to the inferior colliculus, but some longitudinal fibers are still visible on their way to the medial geniculate body just above this. The *tectospinal tract* lies ventrolateral to the medial longitudinal fasciculus (compare with Fig. K). The *rubrospinal tract* is ventral to the brachial decussation. A few cells of the *nucleus of the fourth nerve* may be seen embedded in the dorsal surface of the medial longitudinal fasciculus; the roots of the nerve spring from the lateral aspect of the cell group. The *mesencephalic root of the fifth nerve* is still visible. With it are a few rounded cells, the typical unipolar cells from which this tract springs.

The *pyramids* are much more concentrated than in the preceding section as they have just left the basis pedunculi; in fact, the section on the

left side passes through the edge of the left basis pedunculi. Between the pyramids and the medial lemnisci is a clear space with many rather large cells containing pigment. This is the lower end of the substantia nigra, to be more fully described in the study of the next section.

Section of adult brain stem through rubrospinal (Forel's) and tectospinal (Meynert's) decussation. Before examining the next section of the fœtal brain stem it may be advisable to look at Figure 67, here introduced to show the *rubrospinal (Forel's) decussation,* and the *tectospinal (Meynert's) decussation.* These fibers myelinate rather late and do not show well in fœtal specimens.

Figure 67 is a section stained with Heidenhain's iron hæmatoxylin and passing through the *mesencephalon of an adult at the level of the superior colliculi.* The basis mesencephali shows complete myelination. At its dorsolateral angle are a few isolated bundles of *aberrant pyramidal fibers* and along the medial ventral border are a few more of these aberrant pyramidal bundles. These are destined to control motor nuclei of cranial nerves. Entering the dorsal surface of the basilar portion are many fine fibers which appear to have their origin or end in the substantia nigra. It is just possible that they are axons of substantia nigra cells, and that they may together form the efferent tract of the substantia nigra.

The *substantia nigra* is a sheet of gray matter dorsal to the basis pedunculi and reaching up into the subthalamic region. It gets its name of black substance from its dark color in naked-eye sections; under the microscope this is found to be due to the black pigment in its nerve cells. These are of the large multipolar efferent type. Fine myelinated fibers on its dorsal and ventral surfaces seem to show that it has extensive afferent and efferent connections by fibers which lose their myelin sheaths before they enter it or acquire myelin sheaths after leaving it. Isolated lesions of this substance are almost unknown, though lately cases of paralysis agitans have been described, thought to be due to loss of the cells of the substantia nigra resulting from lethargic encephalitis. It is known that pallidal fibers end in it (S. A. K. Wilson, Fig. 87, F). It is a legitimate guess that it forms part of the mesencephalic tonic mechanism and that it is controlled by the globus pallidus. Its efferent path is unknown.

The tectum (tegmentum) mesencephali. This section (Fig. 67) passes through the *superior colliculi* near their lower bulging, and through the *brachia conjunctiva* after they have crossed and before the red nuclei are beginning to form the relays for the great bulk of their fibers. The section shows a rounded group of myelinated fibers in cross section on

each side of the median plane; the median plane itself is occupied by two distinct groups of decussating fibers. Of these the *ventral decussation,* called *Forel's,* is the decussation of the *rubrospinal tracts.* These tracts begin in the large efferent nerve cells of the red nucleus a little above this, and here cross to become longitudinal, at first ventral to the rounded masses mentioned above; lower down in the brain stem they assume a position ventromedial to the spinal tract of the trigeminal nerve. It will be recalled that in the cord the rubrospinal tract lies ventral to the lateral cerebrospinal (crossed pyramidal) tract. It has been called *Monakow's bundle* and the *prepyramidal tract,* and is the great efferent tract from the cerebellar hemisphere though it only reaches the cord after a relay in the red nucleus.

Meynert's decussation is also seen here. It is the *decussation of the tectospinal tracts.* In Figure 67 delicate fibers sweep in regular curves from the deep surface (stratum profundum) of the superior colliculi round by the lateral and ventral surfaces of the medial longitudinal bundles to a space dorsal to the rubrospinal decussation where they too intercross. The tectospinal tracts have been traced in many sections lower down.

The *medial longitudinal bundles* here together form a V-shaped figure; and the *nuclei of the third* or oculomotor nerves are seen between and dorsal to them. The *fila of the oculmotor nerves,* many of them, pass through the medial longitudinal bundles and sweep in graceful curves through the brachia conjunctiva, after these have intercrossed and before the red nuclei begin to appear. The next section shows their mode of emergence.

The *medial lemniscus* has become triangular on section and lies lateral to the crossed brachia conjunctiva and the red nuclei.

The *lateral lemniscus* forms a finer, more attenuated bundle, lateral to the medial lemniscus. It is on its way to the medial geniculate body, where it will end.

The *inferior brachium,* that is, the brachium of the inferior colliculus, is here seen as a bundle of coarse myelinated fibers on the side of the tegmentum. It consists of cortico-collicular fibers from the superior temporal convolution, the center for hearing, to the inferior colliculus and provides a cortical efferent path from the auditory center to the reflex centers in the inferior colliculi.

The *central tegmental tract* in this section appears as rather scattered bundles of fine fibers in cross section dorsal to the medial lemniscus. Its origin is unsettled. The writer thinks it consists of fibers derived from

the gray matter of the colliculi. It ends chiefly in the superior and inferior olives.

The superior colliculi will be described in the next section.

Figure 68 passes through the *cerebral peduncles and superior colliculi* of a full-term fœtus. On the right side it includes a portion of the *optic tract,* the *lateral and medial geniculate bodies and the thalamus.* As in previous drawings no attempt has been made to indicate nerve cells as they are barely visible with such a low magnification. However, the cells of the *third nucleus* are represented somewhat diagrammatically as regards size but correctly as regards number and grouping. The *pyramidal tracts* are shifting outward to enter the internal capsules. A little lower they would occupy the middle three-fifths of the ventral portion of the basis mesencephali, the medial one-fifth consisting of *fronto-pontine fibers* and the lateral one-fifth consisting of *temporo-pontine fibers* which do not myelinate till some time after birth (compare with Figs. 67, and K and L, long diagram). The *substantia nigra* shows many large pigmented cells not indicated in the drawing.

The *brachia conjunctiva* having decussated at the level of the inferior colliculi are lost in the *red nuclei;* each ends in the red nucleus on the side opposite to that from which it sprang. Some part of each brachium conjunctivum is seen medial to the red nucleus on its way to the lateral nucleus of the thalamus. These fibers also are crossed. On the dorsal surface of the red nucleus are seen *rubrothalamic fibers* uniting the red nucleus with the thalamus; thus an indirect connection is established between the red nucleus and the cerebral cortex (compare Figs. 26 and 87, f). The red nucleus on its lateral side also receives fibers from the globus pallidus.

The *rubrospinal tract* (seen in Fig. 67). From the cells of the red nucleus there starts a new descending tract to the motor nuclei of all motor cranial and spinal nerves. This rubrospinal tract crosses almost immediately to the opposite side (see Fig. 67; compare with Fig. L, long diagram). The rubrospinal tract is the only certain descending tract from the cerebellar hemisphere. There is probably also a descending tract from the vermis by way of the nucleus fastigii, the vestibular nuclei, and the vestibulospinal tract, but this is not proven. The descending cerebellar tract from each hemisphere thus presents two relays and a double crossing; namely, cerebellar cortex to corpus dentatum, corpus dentatum to opposite red nucleus, red nucleus across again to the rubrospinal tract on the side from which the cerebellar connection originally sprang. This

double crossing provides for direct, not crossed, cerebellar connection with lower motor neurons. Dorsolateral to the red nucleus in this section fibers are seen entering it from the globus pallidus of the lentiform nucleus. The red nucleus is normally under control of the globus pallidus which exercises a restraining action on the cerebellar tonic mechanism. There is no previous crossing of lenticulorubral (pallido-rubral) fibers, hence the pallido-rubrospinal influence is crossed. It follows that a lenticular lesion or a lesion of the red nucleus will affect the opposite side of the body from the side of the lesion; in other words, it causes heterolateral or crossed symptoms; while a cerebellar lesion causes homolateral or uncrossed symptoms. The red nucleus sends fibers to the opposite nucleus of the third nerve. No trace of the rubrospinal tract is seen in this section, as the section is a little too high; it was seen in Figure 67. Its position is indicated in Figure L (long diagram).

The *medial longitudinal fasciculus* is here represented by a few scattered bundles by the side of the oculomotor nucleus. The *tectospinal tract* (see Fig. 67, L), of finer fibers and later myelination than the medial longitudinal fasciculus, is here forming by fibers which stream from the deep surface of the superior colliculi (stratum profundum). Many of these fibers intercross dorsally to the aqueduct, and many of them cross ventrally between the red nuclei (Fig. 67). This is not seen in the section, but takes place a little lower (Fig. 67). In the section they appear to form a tract lateral and ventral to the medial longitudinal fasciculus. In the left half of the section many fibers of the stratum profundum seem to enter an area dorsal to the medial lemniscus. The writer believes they here form the *central tegmental tract* described in the preceding section. Its significance is unknown. The *optic tract* sends many terminals (or collaterals) to the superior colliculus (not seen in this section but labeled stratum zonale in Fig. 67); by these fibers as afferents and the tectospinal tract as efferents the superior colliculi coördinate eye and body movements with visual impressions.

Lateral to the red nucleus is the *medial lemniscus.* It is now triangular on section. In Figure 26 it is seen passing into the ventral surface of the optic thalamus where all sensory tracts (except those for the auditory and vestibular senses) end; those represented by the medial lemniscus go to the lateral nucleus, the olfactory tract ends in the anterior nuclei, and the optic tract ends partly in the posterior thalamic nucleus (pulvinar) (see Fig. 68).

As the *medial geniculate body* (Fig. 68) may be regarded as an out-

lying part of the thalamus, the cochlear nerve also may thus be traced to the thalamus; the vestibular is thus the only sensory nerve that has no direct thalamic connections, so far as we know.

Some fibers are here seen also to pass from the medial lemniscus to the superior colliculus. These fibers may form an afferent tract for postural sense to the coördinating mechanism of the colliculus.

Lateral to the medial lemniscus the *lateral lemniscus* is seen ending in the *medial geniculate body,* whence fresh fibers relay the auditory pathway to the superior temporal convolution. Outside the basis pedunculi on the right side the *optic tract* appears to end mainly in the lateral geniculate body, but it also sends fibers to the superior colliculus and pulvinar. The fibers to the pulvinar are well shown; so also are fibers relayed from the pulvinar to the calcarine area of the cerebral cortex (*optic radiation*).

Medial to the lateral geniculate body are *fibers from the optic tract to the medial geniculate body.* These are auditory in function and form a crossed connection between the inferior colliculus of one side and the opposite medial geniculate body passing by way of the optic tract and chiasm (Gudden's commissure).

Oculomotor nuclei and nerves. A few of the many groups of cells which form the *oculomotor nucleus* are seen near the floor of the aqueduct. From the ventral surface of the nuclei the *emerging fibers of the third nerve* flow in bundles which pass round the medial side of the red nucleus to emerge from the medial side of the basis pedunculi (Figs. 23 and 67). No fibers of the third nerve pass through the red nucleus or have any direct connection with it. They pass round its medial side and descend obliquely under it to their superficial origin. If, however, one examines a section passing through the brachium conjunctivum after the crossing, but before it has entered the red nucleus (that is, a little lower down), one will find fila of the nerve passing through the brachium conjunctivum (Fig. 67). Should one get a section through the lower end of the third nucleus, one may find fila of the nerve intercrossing with those of the opposite side. These crossing fibers probably go to the opposite medial rectus for conjugate deviation toward the side from which they crossed. This is the only case of partial crossing of motor cranial nerve roots; the complete crossing of the fourth nerves is also unique.

The oculomotor nucleus is under the control of the opposite pyramidal tract, the cerebellorubral tract, the pallidorubral tract, the vestibular nucleus by way of the medial longitudinal fasciculus, the superior collicu-

lus by way of the stratum profundum, and the opposite, or perhaps the homolateral, sixth nucleus by way of the medial longitudinal fasciculus (compare with Fig. 100).

Above the mesencephalon sections of fœtal brains are unsatisfactory; the chief thing they show is the early myelination of the lenticulorubral bundle which is interesting when one remembers the early appearance of the palæostriatum.

SHORT SUMMARY OF THE NEURONS

(As illustrated in the long diagram, Figures A to M; read the motor and sensory nuclei with reference also to Figures 12 and 13.)

Lower motor neurons. Final common efferent pathway: includes

(*a*) Somatic efferent to voluntary muscles;

(*b*) Splanchnic efferent to involuntary (smooth) muscle of intestines, blood vessels, hairs, etc.; secretory to mucous, digestive, sweat, and sebaceous glands.

Note. Morphologically the so-called splanchnic efferent neurons such as those in the intermediolateral cell column are to be regarded as intercalated neurons between the afferent neurons of visceral sensory nerves and the true visceral efferent neurons of the sympathetic ganglia.

Fig. A. In the *lumbosacral enlargement.* Shows somatic efferent lower motor neurons in the anterior gray column and in the region of the lumbosacral enlargement. They are very numerous and arranged in groups. They correspond with the motor roots of the lumbosacral plexus.

Fig. B. In the *thoracic region.* Shows somatic efferent neurons to trunk muscles, also splanchnic efferent neurons whose cells form the intermediolateral cell column. These last furnish preganglionic fibers to the thoracic and upper lumbar sympathetic ganglia, and to the collateral ganglia in the abdomen.

The intermediolateral cell column extends from the eight cervical to the second lumbar segments of the cord.

Fig. C. *In the region of the brachial plexus,* the anterior gray columns are large and the lower motor neuron cells (somatic) abundant, supplying the limb muscles.

Note. The vessels, hairs, sweat, and sebaceous glands of the arms are supplied with preganglionic efferent neurons from the upper thoracic cord.

Fig. D. *About the level of the second cervical nerve.* Shows lower motor neurons to the neck muscles, and one of the spinal roots of the accessory nerve (to the sternomastoid).

Fig. E. *Through the middle of the pyramidal decussation.* The convexity of the anterior gray column is seen cut off from the central portion by the decussating fibers. It contains motor neuron cells for the first cervical nerve, and behind these the upper end of the spinal nucleus of the accessory nerve whose fila emerge laterally.

Fig. F. *Through the closed part of the medulla oblongata.* Here two columns of somatic motor nuclei are seen.

(*a*) The *hypoglossal nucleus* (twelfth nerve, motor to the tongue) lies close to the central canal and close to the medial sulcus as the medulla oblongata opens. The fila run ventrally and a little laterally, lateral to the medial lemniscus and between the inferior olives and pyramids, between which they emerge.

(*b*) The nucleus ambiguus is the somatic motor nucleus of the vago-glossopharyngeal-accessory group to the pharyngeal and laryngeal muscles. It is dorsolateral to the inferior olive, and in series with the cut-off convexity of the lateral gray column below, and the facial nucleus above. The fila run dorsomedially and then turn round to join the emerging vago-glossopharyngeal group of nerves. These emerge laterally, the lower roots ventral and the highest glossopharyngeal roots dorsal to the spinal tract of the fifth nerve and the intermediate bundles between these points.

(*c*) The *splanchnic efferent nucleus* of the vagus and glossopharyngeal is at first dorsolateral, then lateral to the hypoglossal nucleus in the lower end of the floor of the fourth ventricle, and closed part of the medulla oblongata. The nucleus is in series with the intermediolateral cell column in the thoracic cord. It supplies preganglionic fibers to the smooth muscles and glands of the respiratory and a large part of the alimentary tubes, and inhibitory fibers to the heart.

Fig. G. Shows no efferent nuclei.

Fig. H. Through the *lower end of the pons,* shows:

(*a*) The nucleus of the sixth or abducent nerve, motor to the lateral rectus oculi, lies just lateral to the medial lemniscus in the floor of the fourth ventricle. It is in series with the hypoglossal nucleus. The fila emerge on its medial side; then pass ventrally through the medial lemniscus, corpus trapezoideum, and pyramid, to come to the surface at the lower border of the pons between the pons and the pyramid.

(*b*) The somatic efferent nucleus of the seventh or facial nerve (motor to the muscles of the face) lies deeply in the formatio reticularis, dorsolateral to the superior olive. Its fila pass dorsomedially, form a well-marked ascending rounded bundle dorsomedial to the sixth nucleus. This bundle runs cephalad a short distance, then turns laterally and then ventrolaterally to form a rounded bundle which lies between its own nucleus and the spinal tract of the fifth nerve.

(*c*) Splanchnic efferent nucleus of the seventh nerve, furnishing preganglionic secretory neurons to the submaxillary and sublingual salivary glands and lachrymal gland, and mucous glands of the nose and mouth, is a small-celled nucleus dorsal to the somatic efferent nucleus.

Fig. I. Shows:

(*a*) The upper end of the sixth nucleus.

(*b*) The upper end of the seventh nucleus.

(*c*) The motor (somatic efferent) nucleus of the fifth or trigeminal nerve, motor to the muscles of mastication, the mylohyoid and anterior belly of the digastric. It is in series with the seventh somatic efferent nucleus, but is more dorsal and mainly more cephalad. It lies medial to the large bundle of fibers

which forms the root of the fifth nerve. The fila leave the lateral side of the nucleus to join the emerging root, which runs ventrolaterally and cephalad to emerge from about the middle of the brachium pontis.

Figs. J and K. These figures show the cells and emerging bundles of the fourth or trochlear nucleus (motor to the obliquus superior oculi). This nucleus is in series with the sixth nucleus below and the third nucleus above. It is embedded in the dorsal surface of the medial longitudinal bundle at the level of the inferior colliculi (Fig. K). Its fibers pass laterally, forming a rounded bundle or bundles lateral to the aqueduct and upper part of the fourth ventricle (Fig. K). This runs caudad to the level of the anterior medullary velum (Fig. J), where it turns dorsad into the velum and decussates with its fellow of the opposite side. It emerges from the dorsal surface of the velum (Fig. J).

Fig. L. Shows the nuclei of the third or oculomotor nerves. These nuclei are (*a*) somatic efferent to the levator palpebræ superioris, medial, superior, and inferior recti, and inferior oblique muscles of the eyeball; (*b*) splanchnic efferent to the sphincter iridis and ciliary muscles, and to the lachrymal gland and ciliary body.

(*a*) The somatic efferent nucleus consists of several large-celled groups lying ventral to the aqueductus cerebri at the level of the superior colliculi, and dorsomedial to the medial longitudinal bundles.

(*b*) The splanchnic efferent nucleus is represented by two groups of small cells, notably the Edinger-Westphal nucleus.

The fila of the third nerve emerge from the ventral surface of these nuclei. A few fila intercross with their fellows to join the nerve of the opposite side. The balance of evidence (chiefly physiological) seems to be that these crossed fibers go to the medial rectus of the side opposite to that of their nerve cells for the purpose of conjugate deviation with the lateral rectus on the side of the nerve cells from which these crossed fila sprang. The fila run round the red nuclei or through the brachia conjunctiva after these tracts decussate and emerge just above the pons on the medial side of the cerebral peduncle.

Connections of the lower motor neurons.

(*a*) By the upper motor neurons (pyramidal tracts). From the motor cortex chiefly of the opposite side for voluntary control.

(*b*) By collaterals from sensory nerve roots for short reflexes.

(*c*) With the endogenous neurons of the cord and brain stem for long cord and brain stem reflexes.

(*d*) With the *red nucleus* by the homolateral rubrospinal tract. This comes from the opposite red nucleus and represents:

(1) *Cerebellar influences* which, owing to a double crossing are homolateral.

(2) Through a pallidorubral connection, this cerebellar influence is normally under pallidal control.

(*e*) *Vestibular influences* from Deiters' nucleus:

(1) By way of the medial longitudinal bundle (chiefly crossed) to the third and fourth nuclei.

(2) To the sixth nucleus of the same side probably direct.

(3) By the medial longitudinal bundle chiefly of the same side to neck muscles for turning the head in correspondence with eye movement.

(4) By the vestibulospinal tract of the same side which carries vestibular and probably also cerebellar influences to somatic muscles.

(*f*) Collicular influences from the corpora quadrigemina by way of the tectospinal tract for coördinating eye and body movements with visual and auditory impressions chiefly crossed; also for light reflexes in the iris (both sides).

Higher control of the lower splanchnic efferent neurons.

The collicular control of the iris and ciliary muscle has just been mentioned.

It is known that a nucleus somewhere in the formatio reticularis of the medulla oblongata exerts a homolateral control over the cells of the intermediolateral cell column. Injury to the cervical enlargement of the spinal cord (say between the fifth and seventh segments) causes temporary paralysis of the superior palpebral and orbitalis muscles of Müller, the dilatator iridis and paralysis of vaso constrictor and secretory centers in the intermediolateral cell column. This is homolateral if the injury be one-sided only.

Emotional control from the thalamus and reflex sensory (for example, olfactory) control over splanchnic efferent cord centers reach them by unknown paths.

The upper motor neuron: Voluntary motor and reflex inhibitory.

Fig. M. The cells whose axons form the pyramidal fibers are the large pyramidal cells (Betz cells) in the anterior central gyrus. Perhaps smaller cells give off these fibers in the lower end of this area, as the Betz cell area is very limited at the lower end of the gyrus. (Compare Figs. 90, 91 and 93.) On their way to the internal capsule the pyramidal axons give collaterals to the corpus callosum. The pyramidal fibers descend through the knee and thalamo-lentiform area of the posterior limb of the internal capsule (compare Fig. 87, e). In the internal capsule the fibers to cranial nerves occupy the knee; the neck, arm, and leg fibers, in this order from before backward occupy the thalamo-lentiform portion. In the thalamo-lentiform portion of the capsule motor fibers are mixed with thalamo-cortical sensory fibers (Fig. 87, e).

Fig. L. In the basis mesencephali the pyramidal fibers occupy the middle three-fifths of the basilar portion. Here aberrant fibers to cranial nerves are found (*a*) along the ventral surface of the medial one-fifth; (*b*) a little lower at the lateral side of the substantia nigra (compare Fig. 79, c). Here pyramidal fibers are given to the opposite third nucleus.

Fig. K. Here pyramidal fibers are given to the fourth nucleus, or more likely the fourth nucleus is controlled indirectly from the third nucleus by way of the medial longitudinal bundle.

Fig. J. In the upper end of the pons the pyramidal tract is much broken into bundles. It forms three-fifths of all the longitudinal fibers of the basilar part of the pons and gives collaterals to the nuclei pontis. Aberrant pyramidal fibers to cranial nerves form rounded bundles in the medial margin of the medial lemniscus.

Fig. I. *Middle of the pons.* Here the pyramidal fibers are given to the motor nuclei of both fifth nerves, but especially to that of the opposite side.

Fig. H. *Lower end of the pons.* In the lower pons the pyramidal fibers are concentrated preliminary to the entrance of this tract into the medulla oblongata. Fibers are given:

(*a*) To the opposite sixth nucleus.

(*b*) To both seventh nuclei for the upper face muscles.

(*c*) To the opposite seventh nucleus for the lower face muscles only.

Note. These and other cranial nerve fibers are largely or solely given off from the aberrant fibers.

Fig. G. *In the medulla oblongata* the pyramidal tract is concentrated into a compact bundle on the ventral surface called the pyramid.

Fig. F. Here and a little higher, pyramidal fibers are given off:

(*a*) To the hypoglossal nuclei of both sides, but especially to that of the opposite side.

(*b*) To the somatic motor nucleus of the vago-glossopharyngeal group (nucleus ambiguus). The nuclei of both sides are supplied but especially the opposite nucleus.

Fig. E. *At the pyramidal decussation* the medial three-fourths, more or less, of the pyramidal fibers cross to the opposite side of the cord to form the opposite lateral cerebrospinal tract. The lateral one-fourth continues down the same side of the cord close to the anterior median fissure (Figs. D and C), forming the ventral cerebrospinal tract. This crosses fiber by fiber to supply the lower motor neurons of the opposite side of the cord. It extends down for a variable distance and can often be traced to the sacral segments.

Figs. C to A. *In the cord* the lateral cerebrospinal tracts consist almost entirely of crossed fibers, so that the cortical motor supply of voluntary muscles is almost entirely crossed. However, a few homolateral fibers are usually found scattered in the lateral cerebrospinal tracts, presumably for the supply of muscles such as the intercostals and abdominal muscles, which act habitually in concert on both sides of the body (compare Fig. 79, k to n).

Note. The pyramidal supply of motor nuclei is probably not direct but through intercalated cells as shown in Fig. 69, but not in the long diagram. Nothing is known of cerebral neurons controlling lower splanchnic efferent neurons.

THE SENSORY NEURONS

The lowest sensory neurons for pain, heat, and cold.

Figs. A, B and C. *In the cord* the lowest sensory neurons for pain, heat, and cold are represented by nonmyelinated * axons in the posterior nerve roots. They are derived from the smaller cells of the posterior root ganglia. The peripheral

* It cannot be regarded as settled that the peripheral afferent neurons for pain, heat and cold are nonmyelinated. The writer is disposed to believe that Ranson has shown conclusively that, in the cat, the peripheral afferent neurons for pain are nonmyelinated, and his own anatomical observations dispose him to think that this applies to man.

The fibers for heat and cold (at least the grosser forms) are assumed by the writer to be nonmyelinated on purely theoretical grounds. This is nothing more than a working hypothesis.

processes end in special end organs for each form of sensation; the central processes form the nonmyelinated * fibers of the posterior nerve roots. These enter the posterolateral column, where they probably split into ascending and descending fibers. The most important clinical fact is that they ascend in the posterolateral column for a short distance, fairly constant for each region of the cord and varying between 2 and 6 cm., before they synapse with the neurons which form the relay (second sensory neuron). This synapsis may be direct or through intercalated cells of Golgi type II character in the gelatinous substance.

Second sensory neurons in the cord for pain, heat, and cold. After a short ascending course in the posterolateral column the lowest sensory neurons for pain, heat, and cold are relayed by the neurons of the *posterior spinothalamic tract* (Figs. A, B and C). The cells of this tract are in or just ventral to the gelatinous substance. The fibers immediately cross through the gray substance and in the gray commissure (Figs. A, B and C) and form the opposite *posterior spinothalamic tract.* In the cord this lies medial to the posterior end of the ventral spinocerebellar tract and ventral to the rubrospinal tract. Fibers for pain, heat, and cold are separate but are more or less mingled. In the oblongata this tract is ventromedial to the gelatinous substance (Figs. F and G). In the pons it lies between the superior olive and the gelatinous substance (Fig. H), where it is close to the rubrospinal tract and the ventral spinocerebellar tract. It joins the medial lemniscus in the mesencephalon (Figs. J and K) and with the medial lemniscus enters the ventrolateral thalamic nucleus (Fig. M). Here it is probably relayed by the *third sensory neuron* first to the dorsal part of the lateral thalamic nucleus and from this (*a*) *by a fourth sensory neuron* to the medial thalamic nucleus (essential thalamic organ), where it reaches consciousness and elicits pleasurable or painful sensations; (*b*) *by a variant of the fourth neuron* to the parietal lobe, where probably in the upper lip of the lateral sulcus cortical memories of pain, heat, and cold are recorded and judgments of localization and comparative intensity become possible.

Fig. I. **Lowest sensory neurons for pain, heat, and cold for the face, eyeball, and conjunctiva, nasal and buccal mucosa.** These are in the sensory root of the fifth or trigeminal nerve. The cells are in the semilunar ganglion (Fig. I). The axons are unmyelinated.* Entering the middle of the pons (Fig. I) they form the unmyelinated elements in the spinal tract of the fifth nerve. This descends as low as the upper segments of the cord (Figs. H to D). These neurons are relayed by the trigeminothalamic tract.

Second sensory neurons for pan, heat, and cold for the area of distribution of the trigeminal nerve—trigeminothalamic tract.

Figs. I to D. The lowest sensory neurons for pain, heat, and cold for the face, etc., just described synapse in the gelatinous substance directly or indirectly with the cells of the second sensory neurons. These cross immediately and form the opposite trigeminothalamic tract. Clinical evidence shows that in the medulla oblongata and lower pons this is distinct from the posterior spino-

* See footnote, p. 103.

thalamic tract and lies in the formatio reticularis of the medulla oblongata somewhat nearer the medial lemniscus (Figs. D to G). This also holds good in the lower pons (Fig. I), but in the upper pons and mesencephalon this tract, as well as the posterior spinothalamic tract, lies very near to the outer border of the medial lemniscus (Fig. J). From here on the course of the trigeminothalamic tract is indistinguishable from the other second sensory tracts. Its subthalamic and thalamic relations are similar to those just described. Cortical representation is probably in the lower end of the posterior central gyrus or immediately behind this. There is clinical and experimental evidence that the neurons for pain, heat, and cold in the spinal tract of the trigeminal (fifth) nerve end in the gelatinous substance in such order that successive sensory zones of the face, commencing with the nose and lips and ending with a zone in front of the ear and running to the vertex and chin are represented in order from above downward in the gelatinous substance (Fig. 96, a).

Lowest sensory neuron for muscle-tendon-joint sense as expressed by the sense of active motion and of passive position; the sense of weight as tested by muscular action; postural sense as applied to stereognosis.

Figs. A to C and 69. These neurons are myelinated fibers. Their cell bodies are large cells in the posterior spinal ganglia. Their peripheral processes end in sensory organs in muscles, tendons and joint capsules; their central processes enter the lateral part of the posterior column of the spinal cord where each splits into an ascending and a descending branch. (*Note:* In the lumbosacral region they go through the posterolateral column, and many of them go through the middle of the cap of gelatinous substance.)

The descending branches (Fig. 80, f) are at first close to the dorsal medial border of the posterior gray column and are numerous, sending many collaterals or terminal fibers into the gray matter. Soon they occupy an intermediate position in the posterior column, forming the fasciculus interfascicularis (Figs. 69 and 80, g), and as they descend, approach the posterior median septum. Such descending fibers can be traced from the cervical enlargement as low as the sacral segments (Fig. 81, r to w). They probably serve to coördinate the movements of the fore and hind limbs.

The ascending branches (Fig. 80, d to a) are also at first close to the medial border of the posterior gray column and send many collaterals and terminals into the gray matter. They diminish rapidly as they ascend, but a certain number from each nerve root reach the nuclei of the homolateral dorsal columns in the medulla oblongata (Figs. 80, d to a, 69, and A to F). The fasciculus gracilis consists entirely of long fibers from the lumbosacral plexus; the fasciculus cuneatus contains short fibers, but its long fibers go to the nucleus cuneatus and represent the nerves of the cervico-brachial plexuses. Head believes that the third to the twelfth thoracic nerves have no representation in the nuclei of the fasciculus gracilis or cuneatus.

Second sensory neuron for muscle-tendon-joint sense, etc.

Commencing in the cells of the nucleus gracilis for the leg and nucleus cuneatus for the arm, internal arcuate fibers (Figs. E and F) sweep through the

formatio reticularis, intercross with their fellows in the middle line, and form the opposite medial lemniscus, the fibers for the arm being ventral, those for the leg dorsal (Fig. F). This tract is ribbon-like and lies between the inferior olives in the oblongata. In the lower pons (Fig. H) it is somewhat prismatic in section, is traversed by the corpus trapezoideum, and lies between the pars basilaris and pars tegmentalis close to the middle line (Figs. H and I). In the upper pons and lower mesencephalon (Figs. J and K) it is flattened from before backward, lying between the pars basilaris and pars tegmentalis. In the upper pons or mesencephalon it is joined by the spinothalamic and trigeminothalamic tracts. In the upper mesenchephalon (Fig. L) it is prismatic in section and pushed to the side of the red nucleus, and this is its position (with other tracts for bodily sense) in the subthalamic region. It enters the ventrolateral nucleus of the thalamus, on the opposite side to that in which the lowest sensory neurons lie, the crossing having taken place in the medulla oblongata.

Third sensory neuron for muscle-tendon-joint sense.

There is evidence of a short neuron producing a redistribution and differentiation of the various forms of bodily sense in the dorsal portion of the lateral thalamic nucleus.

Fourth or thalamo-cortical neuron for the muscle-tendon-joint sense complex.

Fig. M. This appears to be chiefly connected with the anterior and posterior central convolutions. In the posterior central convolution (Fig. 90) the sense of position of the thumb seems to be the most sensitive test for the lower end of the gyrus; the sense of position of the little finger and toes for the upper end. In grosser lesions the sense of passive position of the wrist, forearm, and arm are affected, but there is no such definite localization as there is in the case of the motor centers. While the posterior central gyrus is purely sensory and the anterior central gyrus preëminently motor, a limited lesion of the anterior central gyrus causes voluntary paralysis accompanied by numbness and loss of the sense of stereognosis.

Muscle sense complex for the face, masticatory muscles, and tongue.

It seems probable that the trigeminal nerve carries muscle sense for these three groups of muscles, and that the lowest sensory neuron ends in the lateral sensory nucleus of the trigeminal. From this nucleus the relay probably passes to the opposite medial lemniscus, by which the thalamus is reached.

The forms of sensation of special clinical value in the trigeminal sensory area are touch, pressure, pain, heat, and cold, and also taste in the anterior two-thirds of the tongue. The cornea is only sensitive to pain. Vibratory conduction by head and face bones would be inconclusive as regard the fifth nerve because of bone conduction from the temporal by the eighth, ninth, and perhaps the tenth cranial, and by the second spinal nerves. The facial nerve carries fibers for pressure on the face and probably for muscle sense for face muscles. These fibers probably end in the lateral sensory nucleus of the fifth nerve.

Tactile discrimination, or the sense which identifies two points of a compass

as two when they touch the skin simultaneously travels up the same side of the cord to the nucleus gracilis or cuneatus very much the same as muscle sense; but it does not seem to travel up the opposite medial lemniscus, as a lesion of the medial lemniscus in the medulla oblongata may result in loss of sense of passive position for the opposite side of the body without loss of compass sense. In the mesenecephalon and subthalamic region complete interruption of the medial lemniscus causes loss of all forms of sensation for the opposite side of the body. Relays to the dorsal part of the lateral thalamic nucleus and cortex are similar to the last. The site of cortical judgments is probably in the posterior central or superior parietal gyrus.

Vibratory sense as tested by a tuning fork placed against a bone or the skin of the abdominal wall travels up to the posterior columns of the same side of the cord to the bulbar nuclei of the posterior columns, thence by the opposite medial lemniscus to the thalamus. It is represented in the essential thalamic organ and probably in the cortex.

Light touch as tested by a pencil of absorbent cotton and also pressure as tested by graded physical pressure apparently have an inconstant course through the cord.

Light touch travels from the periphery in cutaneous nerves, the writer thinks probably by the fine myelinated fibers in the posterior nerve roots which enter the posterolateral column; these myelinate quite late.

Similar fine myelinated fibers are found in the trigeminal nerve and in its spinal tract.

In cases of lesions of one lateral half of the cord (Brown-Sequard paralysis) touch and pressure are usually not lost on either side of the body below the lesion, indicating that in most cases there is a homolateral route to the medulla oblongata by the posterior column and thence to the thalamus by the opposite medial lemniscus, and also a crossed route by the opposite ventral spinothalamic tract (Fig. 69). In a few cases, however, the route seems to be entirely homolateral by the posterior column. Very rarely is the path entirely crossed.

Again many cases of half lesion of the cord at first lose the sense of light touch and later regain it, while other forms of sensation remain as they were soon after the lesion occurred, so that there may be alternative routes for light touch, one of which may be slowly established when the habitual route is interrupted.

Light touch reaches consciousness in the thalamus, and it is chiefly for the purpose of tactile judgments that it is relayed to the cortex.

In the medulla oblongata the ventral spinothalamic tract joins the dorsal spinothalamic tract (Fig. F); they both join the medial lemniscus higher up.

Pressure sense is carried from the periphery by nerves which travel with the deep motor nerves. On entering the cord, pressure sense appears to follow the same route as tactile sense in each case.

For the distribution of the spinal nerves *pressure pain travels* by deep nerves, but on entering the cord all forms of pain, whether superficial or deep, travel by the opposite posterior spinothalamic tract. Dejerine, however, gives cases

which seem to show that pressure pain travels homolaterally in the cord by the posterior column, crossing in the medulla oblongata.)

In the face, light touch is carried by the trigeminal nerve. After entering the pons it probably has alternative routes by the spinal tract of the trigeminal and opposite trigeminothalamic tract, or by way of the lateral sensory nucleus of the trigeminal and opposite medial lemniscus.

In the face pressure sense and pressure pain at least in some cases may be conveyed by sensory fibers in the facial (seventh) nerve. On entering the brain stem these fibers of the facial for pressure sense and pressure pain probably go to the sensory nuclei of the trigeminal nerve, and thence to the opposite trigeminothalamic tract or medial lemniscus.

Visceral sense in spinal nerves.

This subject is too complex for treatment here. It is discussed briefly in Part II. Speaking generally, visceral pain for the testicle and tunica vaginalis (genito-femoral nerve, L. 1-2), anal canal, urethra and glans penis (pudenal nerve, S. 3, 4, 5), and breast (3rd, 4th, 5th, and 6th intercostals), travels up by the peripheral nerves indicated, reaching corresponding cord segments by the posterior roots.

For abdominal and thoracic organs visceral pain travels by nerves which pass through the sympathetic ganglia without interruption, join spinal nerves by white rami communicantes, and so enter the spinal cord. The segments of the spinal cord that receive afferent nerves from the individual viscera are probably those which give rise to referred visceral pain as indicated in Figure 94.

Thus sensory nerves from the uterus and rectum, prostate and neck of the bladder probably enter the first three sacral segments. So also the ovary (like the testis and tunica vaginalis testis) is connected with the first and second lumbar segments; the kidney, ureter, and upper part of the bladder with the twelfth thoracic and first lumbar segments; the colon with the eleventh and twelfth thoracic; the small intestine with the ninth and tenth thoracic; the stomach with the sixth and seventh thoracic; the œsophagus with the fourth to the sixth thoracic; the breast with the fourth to the sixth thoracic; the heart with the second to the fourth thoracic; the diaphragm, gall bladder and bile ducts with the third to the fifth cervical (phrenic nerve). These connections can partly be demonstrated anatomically, and are partly postulated from the clinical observation of the localities of referred pain and tenderness in painful affections of the various organs.

The second sensory neurons for visceral sense.

It is known that the sense of visceral pain travels with other forms of pain by the posterior spinothalamic tract. The spinal tracts are not known by which travel other forms of visceral sense, such as the desire for defecation and the sense of fullness of the bladder; one may suppose, however, that they travel like somatic touch and pressure. It is certain that all forms of visceral sense set up spinal reflexes, and that all reach consciousness in the thalamus; there

is reason to believe that pelvic reflexes are controlled in the anterior central portion of the paracentral lobule (Fig. 93).

Visceral sensory branches of the vagus (Fig. F).

While gastrointestinal pain is carried by visceral branches of spinal nerves, it is probable that the sense of hunger, nausea, comfortable satiety, overfullness of the stomach, are functions of the afferent branches of the vagus. These appear to end in the dorsal nucleus of the vagus (Fig. F). This nucleus is regarded as mixed in character, containing visceral efferent cells, and also the cells whose axons relay visceral afferent impressions. The paths of visceral sense in the brain stem are unknown.

Gustatory sense (Fig. F).

The gustatory nerves are the glossopharyngeal for the posterior third of the tongue, the pillars of the fauces, and the soft palate, and the chorda tympani for the anterior two-thirds of the tongue.

Glossopharyngeal. The taste fibers of this nerve have their nerve cells in its two ganglia. The central axons enter the fasciculus solitarius (Fig. F). In the nucleus of the fasciculus solitarius, which all neurologists regard as the gustatory nucleus, a relay must take place, but the course through the tegmentum of the pons and mesencephalon is unknown. The cortical representation of taste is not definitely known, but is supposed to be in the neighborhood of the olfactory area in the anterior hooked extremity of the hippocampal gyrus.

The nerve of taste for the anterior two-thirds of the tongue is the *chorda tympani*, a branch of the facial. Its ganglion cells are in the geniculate ganglion of the facial. It enters the brain stem between the facial and auditory where it is known as the nervus intermedius. It joins the fasciculus solitarius.

(*Note:* There are other theories about the central connections of the chorda tympani, but this is probably the correct one. For the others, books on nervous diseases may be consulted.)

The eighth nerve.

This nerve consists of two parts, the vestibular nerve from the vestibule and semicircular canals, conveying equilibratory sense, and the cochlear nerve, which is the nerve of hearing and comes from the cochlea. The vestibular nerve is so closely related to the cerebellum that it will be discussed with the cerebellar afferent paths, and here the nerve of hearing only will be considered.

Sense of hearing: Lowest sensory neuron. The cochlear nerve conducts auditory impressions from the cochlea which is the organ of hearing. Its ganglionic cells are the bipolar nerve cells in the spiral canal of the cochlea. In this ganglion in vertebrates the cells retain in adult life their original embryonic bipolar form. The central axons enter the upper end of the medulla oblongata lateral and caudal to the vestibular nerve (Fig. 102). Here it divides into two sets of fibers, one of which ends in the ventral cochlear nucleus, ventral and ventrolateral to the restiform body (Figs. 60, 61 and 102). The other

winds round the lateral aspect of the restiform body in the region of the lateral ventricular foramen and ends in the dorsal cochlear nucleus (Fig. 102).

Sense of hearing: Second sensory neuron. The second sensory neurons for hearing commence in the cells of the ventral and dorsal cochlea nuclei (Fig. 102), form the corpus trapezoideum (Figs. 102, 62, H and I), the fibers of which partly decussate, are connected with the superior olives, chiefly that of the same side, and take a cephalad direction in the neighborhood of the superior olive, forming the lateral lemniscus. The fibers from the dorsal nucleus form the striæ medullares (Fig. 102) send fibers to both superior olives, and join the corpus trapezoideum chiefly in two groups of fibers. One group winds round the dorsal and then the medial side of the homolateral restiform body to join the corpus trapezoideum homolaterally; the other crosses the floor of the fourth ventricle, dips into the median sulcus and joins the corpus trapezoideum on the side opposite to the nucleus of origin (Fig. 102).

The *corpus trapezoideum* (Figs. H and I) derived as described from axons belonging to the cochlear nuclei, forms a well marked sheet of deep transverse fibers of the pons, lying between its basilar and tegmental portions. It is found just as the medulla oblongata joins the pons. It is traversed in its medial third by the medial lemniscus (Fig. H), and its fibers are interrupted by the superior olivary nuclei and other groups of nerve cells.

The *superior olive* (Figs. 102 and H) is a group of nerve cells situated in the substance of the corpus trapezoideum receiving collaterals from the cochlear nuclei of both sides chiefly of the same side. It lies ventromedial to the facial nucleus in the lower end of the pons. Many of its axons continue in the corpus trapezoideum and are prolonged into the lateral lemniscus of the same or opposite side (Fig. J). It is connected with the homolateral abducent nucleus (Figs. I and 102) by fibers which run dorsally, forming the *peduncle of the superior olive.* Thus a short reflex path is formed for lateral deviation of the eyes toward the direction from which a sound appears to come (homolateral ear).

Lateral lemniscus. The lateral lemniscus, which is the upward continuation of the corpus trapezoideum, is concentrated at the upper end of the pons (Fig. J) as a new longitudinal tract between the tegmental and basilar portions of the pons, distinguished from the medial lemniscus by its finer fibers and more lateral position. It soon shifts dorsolaterally to the surface of the brachium conjunctivum, where a nucleus appears among its fibers called the nucleus of the lateral lemniscus. The function of this nucleus is unknown. At the level of the inferior colliculi a great part of the lateral lemniscus passes backward to the inferior colliculus, its fibers enclosing and ending in this nucleus (Fig. K). The rest of the lateral lemniscus ends a little higher in the medial geniculate body (Fig. L), from which springs the third or geniculocortical relay. (This is the third relay, if one disregards the interruptions taking place in the superior olive and in the nucleus of the lateral lemniscus, and these may not be complete interruptions.)

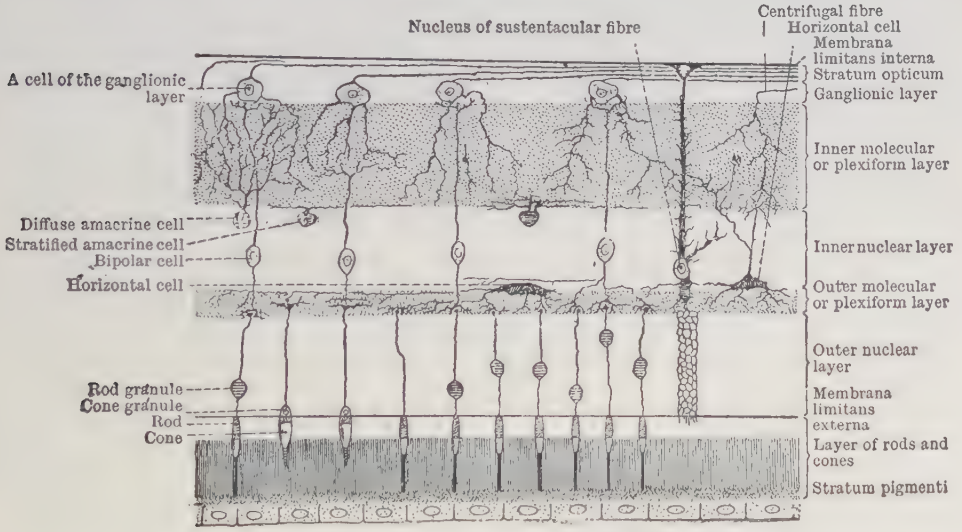
The third sensory neuron for hearing. The cells of the medial geniculate body give off axons which form the auditory radiation (Fig. M). This connects the medial geniculate body with the posterior third of the superior temporal

convolution and the anterior transverse temporal gyrus (Figs. 90 and 92). This is the auditory portion of the cerebral cortex. Each medial geniculate body and superior temporal gyrus is connected with both ears.

While the medial geniculate body and superior temporal gyrus are auditory in function, the inferior colliculus in which many of the fibers of the lateral lemniscus end, is not sensory but is a reflex center for coördinating eye and body movements with auditory impressions. It is connected with the temporal region of the cortex cerebri by corticifugal, but not by corticipetal fibers, and with the nuclei of the eye muscles and spinal cord by fibers of the tectospinal system (Fig. K).

The tracts for the sense of vision.

The *optic nerve* (so called) and *retina* are outgrowths from the forebrain. Of the retina (see subjoined figure) the rods and cones with the nuclei in the outer nuclear layer and terminal arborizations in the outer molecular layer are special end organs. The bipolar cells with their peripheral and central processes form collectively the equivalent of the peripheral nerve of sight (lowest sensory neuron). These end by arborizing with the dendrites of the cells of the ganglionic layer, and these cells are collectively to be regarded as the terminal nucleus of



A plan of the retinal neurons. (After Cajal.)

the lowest sensory neurons for sight and the nucleus of origin of the second sensory neuron. Their axons form the centripetal fibers of what is usually called the optic nerve, but the optic nerve is really the second sensory nerve tract for the sense of sight.

Centripetal fibers of the optic nerve (Fig. 105). The fibers from the temporal half of each retina pass at the optic chiasm into the homolateral optic

tract, while the fibers from the nasal half of each retina decussate with their fellows in the chiasm; thus the left optic tract is composed of fibers from the left halves of both retinæ, and the right optic tract consists of fibers from the right halves of both retinæ. The maculæ are represented by both crossed and uncrossed fibers. As light rays cross in the lens it follows that the right halves of the fields of vision of both eyes are represented in the left optic tracts and vice versa. If now for the sake of clearness we confine our attention to the left optic tract, the visual fibers of this tract end in the lateral geniculate body, the superior colliculus, and the pulvinar of the thalamus (Figs. 105, 68, and L). Of these three centers the lateral geniculate body is the subcortical visual center, the pulvinar is not visual but is probably associated with the pleasurable or painful elements in light perception, and the superior colliculus is a reflex center for pupillary reaction, accommodation, and eye and body movements.

Third sensory neuron for vision. Optic radiation. The cells of the lateral geniculate body and pulvinar give off axons which form the optic radiation (Fig. 105). These fibers flow round the posterior horn of the lateral ventricle and end in the left area striata, which lies in the walls of the calcarine fissure, and in the cuneate and lingual lobes (Figs. 90 and 93). Fibers from the upper left quadrants of both retinæ radiate to the upper part of this area, and fibers from the lower left quadrants of both retinæ end in the lower part. The macular fibers of each eye go to both hemispheres.

Thus the left optic tract, left lateral geniculate body, left optic radiation, and left area striata all receive light impressions from the *right halves of the fields of vision* of both eyes; and of these fields the upper quadrants are received by the lower portions of the visual areas. The pulvinar and superior colliculus are connected with the occipital cortex by corticothalamic and corticocollicular fibers, and it is probable that the efferent fibers in the optic tracts and optic nerves which go to the inner molecular layer of the retinæ come from the superior colliculi.

Visual reflex tract. Certain of the cells of the superior colliculi give off axons which form the stratum profundum album (Fig. L). There is a dorsal decussation of fibers from the opposite colliculi. These fibers flow round the central gray matter and under the medial longitudinal bundles, decussate with each other between the red nuclei in Meynert's decussation (Fig. 67) and form the tectospinal tracts. In the mesencephalon, pons, and medulla oblongata it is ventral to the medial longitudinal bundles and in the cord goes to the ventral white columns lateral to the ventral median fissure. This tract is connected with motor nuclei for eye, neck, and body muscles, and serves to coördinate reflex pupillary, accommodation, and ocular and body movements with visual impressions. The tectospinal tract is shown in circles in Figure 69 and Figures J to B.

Olfactory paths. For the present purpose it is sufficient to remember that the peripheral nerve of smell is represented by the aggregated nerve filaments whose hair-like endings in the olfactory area of the nasal mucosa (a small area in the upper part of the septum and superior conchæ) are the perceptive endings; the nuclei of these cells in the nasal mucosa represent the ganglion, and their central nonmyelinated processes which pass through the cribriform plate repre-

sent the lowest olfactory path. These end in glomeruli in the olfactory bulb, where they synapse with dendrites of the mitral cells. The centripetal axons of these mitral cells in the olfactory tract represent the secondary pathway. The connections of this are exceedingly complex, but sooner or later it reaches the anterior extremity of the hippocampal gyrus (Fig. 93), which in man is the cortical center for smell. The connection is probably bilateral. The anterior nucleus of the thalamus probably represents the thalamic center for smell and is responsible for pleasurable or unpleasant reactions to this form of sensory stimulus. In man the sense of smell is more or less rudimentary.

PART II

SUMMARY OF THE ANATOMY AND PHYSIOLOGY OF THE NEURONS

PART II

ANATOMY AND PHYSIOLOGY OF THE NERVE TRACTS

SUMMARY OF THE NERVES AND TRACTS

Note. Here begins the summary of a lecture course on Applied Neurology to Junior students delivered by me in the University of Texas for the past ten years. There is necessarily considerable repetition of the preceding summary of the anatomy of the nerves and tracts as it was arranged for Sophomore students to conclude their laboratory study of the nerve tracts in the Sophomore neurology.

After the student has become acquainted with the neurons and tracts in detail, he should study the following summary with the help of the long diagram. Every detail should be studied with the pictures, especially the long diagram, that the situation of each tract may be firmly impressed on the mind. Only in this way can he visualize the possible nervous lesions which may account for the symptoms observed in clinical cases. The capital letters refer to the figures in the long diagram, the numerals to the figures in the Atlas.

PRELIMINARY NOTE

The *lower or peripheral efferent neurons* of the cord and brain stem may be divided into *somatic efferent* to striped muscle, and *splanchnic efferent* to smooth muscle and viscera. Now the latter are not morphologically or physiologically in the same class with the former. The peripheral efferent neurons to striped muscles have their cell bodies in the anterior gray columns of the spinal cord or in the brain stem; the corresponding neurons for viscera, blood vessels, skin glands, and erector muscles of the hairs have their cell bodies in the sympathetic ganglia, or are imbedded in the viscera themselves.

The "splanchnic efferent neurons" in the spinal cord and brain stem are really neurons intercalated in the reflex arc between the peripheral and afferent and peripheral efferent neurons. Reference to the diagram will make the interrelations clear.

On the right side of the figure the visceral reflex arc is shown; on the left side, the somatic reflex arc. In each there is an intercalated neuron. In the visceral reflex arc the efferent neuron has migrated into the sympathetic ganglia, or, as in the whole parasympathetic system, still more distally into the viscera, and the visceral intercalated neuron assumes falsely the appearance of a true peripheral efferent neuron. On the other hand, in the case of the somatic reflex arc, shown on the left side of the diagram, the whole intercalated neuron and the cell body of the peripheral

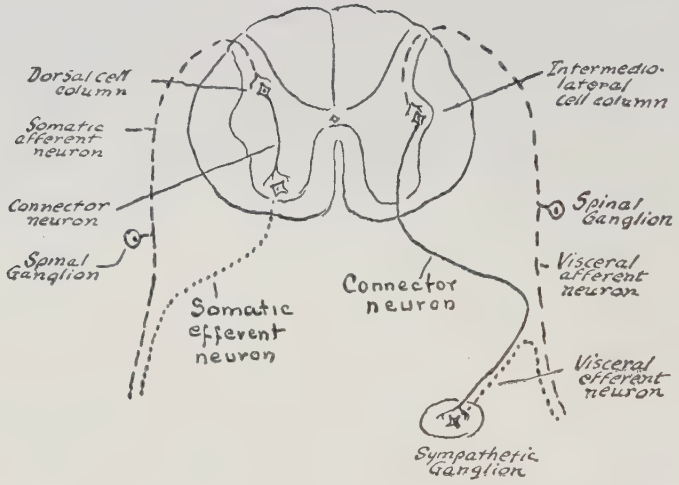


Diagram showing the relations of the neurons in the somatic and splanchnic reflex arcs. (From Langdon Brown.)

efferent neuron remain within the central nervous axis. The common phraseology will still be retained, but the difference in meaning must be remembered. The intermediolateral cell column of the sympathetic system, and the visceral nuclei of the third, seventh, and tenth cranial nerves (together forming the parasympathetic nuclei in the cord and brain stem) belong to the intercalated cell system in the reflex arcs involved.

In considering the salient points in the *Physiology* of the nervous system that have the most obvious bearing on the interpretation of the symptoms of nervous diseases, one must first direct attention to the lowest *reflex arc*. Through this all higher nervous mechanisms must act.

This lowest reflex arc is illustrated in Figures 75 and 76, and can be traced out in Figures 69 and 70; while a glance at Figures 2, 4, and 53 readily discovers possible paths in the cord forming this arc. A simple reflex is illustrated by the withdrawal of the hand when the finger has unwittingly touched an overhot plate. Yet, though the act seems simple,

a very complex mechanism is required. At the least there is involved one or several afferent neurons for pain, an intercalated cell group, and efferent neurons supplying the necessary muscles.

A shade more complex is the mechanism needed for the scratch reflex of a dog (Fig. 76) where tickling a fore-shoulder causes scratching with the hind leg. Here long association fibers between the cervical and lumbosacral enlargements of the cord are necessary. In addition, secondary higher reflexes are required for the maintenance of equilibrium, involving reflex action of the cerebellum and basal ganglia, with their afferent and efferent tracts. Superimposed above all reflex arcs for purposes of volitional control is the voluntary motor or pyramidal system and it must be remembered that voluntary efforts are not attained through direct action of the pyramidal neuron on the lower motor neuron, but rather by its use of all the subsidiary reflex systems.

THE MOTOR NEURON

The lower motor neuron is the only pathway for kinetic efferent impulses to striped muscle.

Evidence is accumulating that all or most skeletal muscles have a sympathetic unmyelinated nerve supply for the purpose of maintaining plastic (that is, postural) tone. These neurons come from the cells of paravertebral sympathetic ganglia in the cervico-brachial and lumbosacral regions, reach the muscles through gray rami communicantes to the spinal nerves, and are controlled by white rami communicantes from the spinal cord. The higher control is uncertain and the whole subject needs further investigation.

In **Figure A, lumbar enlargement** (compare Figs. A, 4, 8, and 9) the grouping of the cell bodies is indicated diagrammatically as well as the emergence of their axons in several bundles. The axons end in striped muscles. The abundance and the grouping of the cells as seen in Figure 8 are characteristic of these segments, and are dependent on the necessity for supplying the muscles of the lower extremity. In the second and third, or third and fourth sacral segments, groups of small cells are situated posteromedial to the posterolateral large-celled groups. They supply visceromotor preganglionic fibers to the pelvic viscera. Their axons leave the cord with the somatic motor nerve roots.

Thoracic region, Figure B (compare Figs. 3, 7, and 54). In the thoracic region in addition to the usual lower motor somatic neurons there is a column of visceromotor, or better, splanchnic efferent, neurons called the intermediolateral cell column. This column extends from the first

thoracic to the second lumbar segments, inclusive. Its axons form the white efferent rami communicantes (preganglionic fibers) to the sympathetic or autonomic nervous system. It should be remembered that these are really intercalated neurons in the visceral reflex arc (page 117).

Cervical enlargment. Nothing of special interest is noted here except the abundance and grouping of the cells necessary to supply the arm muscles (compare Fig. C with Figs. 2, 6 and 55).

Upper cervical segments. From the third cervical segment upward a special cell column, somewhat centrally placed, gives rise to the emerging axons of the *accessory nerve.* This cell column can be traced as low as the sixth cervical segment; above, it is continuous with the nucleus ambiguus. In some sections the emerging fila of the accessory nerve can be seen passing first dorsally, then laterally (Figs. D, 1, and 56) to emerge from the cord in bundles behind the ligamenta denticulata.

THE LOWER MOTOR NEURONS OF CRANIAL NERVES

The twelfth or hypoglossal nerve is motor to the tongue (Figs. F, 10, 12, 14, and 59). Its cell bodies form a column of cells extending from just above the pyramidal decussation to the level of the striæ medullares (Fig. 12). The nucleus is at first ventrolateral, and then lateral to the central canal (Fig. 14, 59, F). In the open part of the medulla oblongata it lies in the floor of the ventricle near the median sulcus beneath the trigonum hypoglossi (Fig. 11). It is a typical large-celled somatic motor nucleus. The axons are gathered into bundles which pass ventrally lateral to the medial lemniscus and pyramid, and medial to and partly through the medial side of the inferior olive. They emerge along the groove between the inferior olive and the pyramid (Fig. 10).

The nucleus of the sixth or abducent nerve (motor to the lateral rectus oculi) lies in longitudinal series with the twelfth, fourth, and third nuclei (Fig. H; compare Figs. 12, 13, 62, and 63). It is a rounded column of typical lower motor neuron cells which lies in the floor of the fourth ventricle close to the median sulcus, just above the striæ medullares, and subjacent to the prominence in the floor called the colliculus facialis (Fig. 11). It is just ventrolateral to the medial longitudinal fasciculus, with which it is intimately connected. It has a very marked special connection with the superior olive (Fig. H; compare Fig. 63), by which a special auditory reflex arc is provided. The axons leave its medial border and pass ventrally in bundles through the medial lemniscus, corpus trapezoideum and pyramid, to emerge in the groove between the pons and

medulla oblongata near the middle line (Fig. 10). A ventral nucleus of the sixth nerve is described by Van Gehuchten in the rabbit and is readily identified in man. It is unnecessary for our purpose to pay further attention to it.

The fourth or trochlear nerve is motor to the superior oblique muscle of the eyeball (Figs. J and K; compare Figs. 12, 22, 18, 66, and 64). The nucleus consists of a short column of cells in series with the sixth and third nuclei. It lies in the mesencephalon under cover of the inferior colliculi, and is imbedded in the surface of the medial longitudinal bundle (Fig. 66) with which it has intimate connections. Its axons run laterodorsally and form one or two rounded bundles which descend along the side of the aqueduct and upper end of the fourth ventricle to the lower border of the inferior colliculi. Here they intercross in the anterior medullary velum, emerging from this at the medial border of the brachium conjunctivum; (compare Figs. K and J). The decussation is complete, and the course of the fibers is altogether exceptional.

The third or oculomotor nerve. The third or oculomotor nerve is *motor* (somatic efferent) to the levator palpebræ and all the extrinsic eye muscles except the lateral rectus (sixth nerve) and superior oblique (fourth nerve); it is also *visceral efferent* (splanchnic preganglionic neurons) to the sphincter of the iris and ciliary muscle. The nucleus consists of a series of distinct groups of cells in the ventral gray matter of the aqueductus cerebri (Fig. L; compare Figs. 68, 10, and 12). The column of cells is 5 to 6 mm. long and lies at the level of the superior colliculi (Fig. 12). A small-celled nucleus gives rise to the *splanchnic efferent* fibers for the intrinsic eye muscles; its fibers are relayed in the ciliary ganglion. The nucleus lies imbedded between the two medial longitudinal bundles from which it receives association fibers connecting it with the vestibular and abducent nuclei, and perhaps with the superior olive. It probably sends association fibers by way of the medial longitudinal bundle to the nucleus of the fourth or trochlear nerve.

Many bundles of axons leave the ventral surface of the nucleus. They run around and between the red nuclei, the lower fibers passing through the brachium conjunctivum, and they emerge on the medial side of the basis mesencephali (Figs. 23, 68 and 10). Some of the axons are crossed, and it may be that these crossed axons are destined for the medial rectus opposite to their origin so as to correlate the action of the medial rectus with the opposite lateral rectus (supplied by the sixth nerve) for purposes of conjugate deviation.

It has seemed best to take the twelfth, sixth, fourth, and third cranial nerves together because they are all so distinctly in series. The motor nuclei of the accessory, vagus, glossopharyngeal and facial nerves make up a second series. These nuclei form a discontinuous column of cells deeply and somewhat laterally placed in the cord, and in the formatio reticularis of the oblongata and lower pons. Their splanchnic efferent nuclei form a similar interrupted column more dorsally placed. The vagus, glossopharyngeal, and facial are mixed nerves and their sensory roots and terminal nuclei will be described later.

The eleventh or accessory nerve (Figs. D and E; compare Figs. 56, 57, 58, also 10 and 12). The eleventh nerve consists of a spinal portion for the supply of the upper parts of the trapezius and sterno-mastoid muscles, and a cerebral portion which forms the motor fibers of the superior and inferior laryngeal nerves for the supply of the muscles of the larynx.

The spinal part of the eleventh nerve, illustrated in Figures D and E (compare Figs. 56, 57, 58, and Figs. 12 and 13) arises from a column of cells extending as low as the fifth cervical segment. The column is behind the other cells of the anterior gray column, as seen in the figures. At the level of the pyramidal decussation it lies in the detached part of the anterior gray column (Figs. 58 and E). The axons form bundles which pass dorsally, then laterally, emerging as a series of roots just behind the denticulate ligament to join an ascending trunk. Part of the ascending portion of the nerve is imbedded in the gray matter dorsal to the nucleus.

The cerebral part of the accessory nerve arises from the lower part of the nucleus ambiguus (Figs. 59 and 14). Those fibers of the accessory nerve which supply the laryngeal muscles are in series with those fibers of the vagus which supply the pharyngeal and palate muscles and with those of the glossopharyngeal which supply the stylopharyngeus; indeed, it is convenient to regard the cranial part of the accessory nerve as the somatic motor root of the vagus. The nucleus is the nucleus ambiguus (Fig. F; compare Fig. 59). It is in series with the facial (or seventh) nucleus. The nucleus ambiguus is a column of large multipolar cells, imbedded deeply in the reticularis grisea, between the dorsal accessory olive and the gelatinous substance (Fig. 14), and is best seen in the lower open part of the oblongata. The axons spring from its dorsal surface, run dorsally, then curve round laterally and ventrally to join the emerging bundles of the glossopharyngeal-vagus-accessory group (Figs. F and 14).

Of this group the lower fibers run ventrolaterally, ventral to the spinal tract of the fifth nerve (Fig. 58), while the upper fibers run through or dorsal to the spinal tract of the fifth (Fig. 59).

The **splanchnic efferent nucleus of the vagus and glossopharnygeal** (Figs. F and 14) consists of a column of medium-sized cells in the floor of the oblongata, at first latero-dorsal and then lateral to the hypoglossal nucleus, extending as high as the striæ medullares (Figs. 11 and 12). The axons form bundles which run ventrolaterally through or dorsal to the spinal tract of the fifth nerve, and are accompanied by the sensory vago-glossopharyngeal roots. These splanchnic efferent neurons form the preganglionic fibers (cephalic parasympathetic) for the parotid gland and for the glands of the back of the tongue, the larynx, pharynx, and entire alimentary and respiratory tracts; they also furnish the splanchnic efferent fibers for the alimentary and respiratory unstriped muscles, and the vagus gives in addition fibers to the heart. The sensory roots of the vagus and glossopharyngeal nerves will be described on page 137.

The seventh or facial nerve (Figs. 10, 12, 13, 24, H, 60, 61, 62 and 63). The seventh or facial nerve, which supplies the muscles of the face, and the stapedius, has its somatic efferent nucleus placed deeply in the formatio reticularis in series with the nucleus ambiguus. This nucleus is best seen at the level of the corpus trapezoideum (Fig. H, 60, 61 and 62), where it lies dorsolateral to the superior olive. There are three ventral groups of cells and a dorsal group differing somewhat in the size of their cells, but all typically somatic efferent in character. From this extensive nucleus the axons pass dorsomedially around the sixth nucleus (Figs. 24, 60, 62) to reach a longitudinal bundle in the floor of the oblongata underlying the colliculus facialis (Fig. 11) in the dorsal angle between the sixth nucleus and medial longitudinal bundle. After a short course in an ascending direction this compact bundle runs laterally over the sixth nucleus (Fig. 24) and then bends ventrolaterally medial to the spinal tract of the fifth nerve and gelatinous substance and lateral to the upper end of its own nucleus to emerge at the lower edge of the pons (Fig. 10).

The splanchnic efferent nucleus of the facial nerve furnishes preganglionic (parasympathetic) fibers for the lacrymal gland by way of the great superficial petrosal nerve and sphenopalatine ganglion, and preganglionic salivary fibers for the submaxillary and sublingual glands by way of the chorda tympani nerve and submaxillary ganglion. This nucleus is a small rounded group of nerve cells in series with the splanchnic

vago-glossopharyngeal nucleus. It is lateral to the sixth nucleus and just medial to or among the fibers of the emerging root of the seventh nerve as it turns ventralward. It is dorsal to the somatic nucleus from which it is separated by a considerable interval (Figs. H, 62 and 63).

The facial nerve has a few cutaneous sensory fibers for the external auditory meatus and a gustatory root for the anterior two-thirds of the tongue which forms the sensory portion of the chorda tympani, to be described on page 156.

The motor nucleus of the fifth nerve (trigeminal) is purely somatic and supplies the muscles of mastication (Fig. I; compare Figs. 16, 63, 10, 12, and 13). It is in the same sagital plane with the upper end of the somatic facial nucleus, but is more dorsally placed (Figs. I and 63), so that it lies near the floor of the fourth ventricle just lateral to the superior fovea (Fig. 11). It is rather a large group of multipolar nerve cells, from the ventral and lateral surface of which the axons emerge to form a large bundle running ventrally and a little laterally to the point of emergence from the middle of the brachium pontis (Fig. 10). It is joined by emerging fibers from the mesencephalic root. The function of these last is unknown, and they will not be described further. The sensory root of the fifth nerve will be described on page 139.

Higher control of lower motor neurons. These somatic motor nuclei are all the somatic motor nuclei of the cranial nerves. They all receive voluntary activating or inhibitory control from the pyramidal tract. The pyramidal supply of the nuclei to the extrinsic muscles of the eyeball (recti and obliqui; third, fourth, and sixth nerves) and of the nuclei to the lower facial muscles is entirely crossed; while the pyramidal supply of the upper facial muscles (orbicularis oculi, frontalis, corrugator supercilii), and of the muscles of mastication, as well as the larynx, pharynx and tongue is partly homolateral but mainly crossed. In the trunk, muscles which act habitually together, as the intercostal and some spinal muscles, get a partly homolateral pyramidal supply, but the main pyramidal supply for spinal nerves is crossed (see upper motor neuron). All the lower motor neurons receive collaterals from corresponding sensory nuclei. All receive impulses from distant parts of the brain stem and cord by association fibers; and indirectly from the lentiform nucleus, as well as from the red nucleus, cerebellar nuclei, and vestibular nuclei by tracts to be described later (Fig. 100).

The third, fourth, and sixth nuclei have special connections with the vestibular nerve by way of Deiters' nucleus and the medial longitudinal

bundle; with the cochlear nerve by way of the superior olive and its peduncle; with the cerebellar nuclei either by way of the rubrospinal tract or by way of Deiters' nucleus and the medial longitudinal bundle; and with the corpora quadrigemina either directly or by way of the medial longitudinal bundle. By this route also they are intimately connected with each other by association neurons.

Higher control of lower splanchnic efferent neurons. All that is known of the higher control of lower splanchnic efferent neurons can be expressed in the brief statements which follow.

The centers for accommodation and for contraction of the pupil in the third nerve nucleus are probably under control of the superior colliculi, either directly by way of the stratum profundum or indirectly by way of the medial longitudinal bundle. The *cortical control* is in the area striata round the calcarine fissure, in the posterior end of the middle frontal gyrus, and perhaps in the angular gyrus.

The pupil dilating center, together with the center controlling the orbitalis and superior palpebral muscle in the eighth cervical and first thoracic segments of the spinal cord is probably controlled by the superior colliculus by way of the tectospinal tract.

The splanchnic efferent nuclei of the intermediolateral cell column are controlled by cells in the formatio reticularis of the medulla oblongata by way of a homolateral bulbospinal tract in the anterolateral column. Nothing is known of the higher control of this nucleus. The fact that there is undoubtedly a thalamic control, at least over the vasomotor function of the nuclei is demonstrated by such phenomena as blushing, sweating and cardiac acceleration, produced by emotion.

PHYSIOLOGY OF THE LOWER EFFERENT NEURONS

The *lower somatic efferent neuron* forms the only efferent tract to striped muscles. All nervous influences which reach the muscles, whether they are spinal reflexes or cerebellar, vestibular, or striate reflexes, or whether they are cortical activating or inhibiting stimuli, all must reach them through the lower somatic efferent neuron (Fig. 100). Therefore, destruction of this neuron anywhere from cell body to muscle plate causes loss of all nervous control over the muscle, with (*a*) paralysis, (*b*) flaccidity, (c) loss of reflexes, (d) electric reaction of degeneration in the muscles, and finally histological degenerative changes, hyaline, fatty, and fibrous, in the muscle fibers.

(*a*) The *paralysis* is complete; all movements, voluntary, reflex and

associated, are lost. This is due to severing the connection between the muscle fiber and the cell body of the lower motor neuron, without which no efferent impulse, voluntary or otherwise, can be transmitted.

(*b*) *Flaccidity.* In health all voluntary muscles are in a condition of tonic contraction. This depends on a constant flow of entering sensory impressions through the sensory nerves. These, mostly through intercalated cells in the spinal cord, send impulses to the lower motor neuron. By these lower motor neurons the entering stimuli are converted into an equally constant flow of outgoing activating stimuli to the muscles, thereby maintaining muscle tone. The loss of the lower motor neuron breaks the reflex arc by which the steady stream of stimuli is maintained, and the paralyzed muscle is flaccid.

(*d*) The integrity in structure of a muscle depends on its vital connection with the lower motor neuron supplying it. The reason for this is not entirely clear; it cannot be explained on the basis of disuse only. The fact remains that separation from the lower motor neuron is invariably followed by degeneration, of which the earliest clinical evidence is the peculiar electric reaction known as the reaction of degeneration. Microscopic structural changes soon follow.

Each of the limb muscles appears to be supplied by at least two segments of the spinal cord. Parts of the muscle may degenerate while others remain normal according to the extent of the lesion to the nerve supplying it.

Examples of lower motor neuron paralysis due to various types of lesion are illustrated by figures 151 to 166. Figures 160 and 161 show the disappearance of the anterior horn cells in a case of chronic progressive anterior poliomyelitis, a disease characterized by slowly progressive degeneration of these cells. Figure 162, a drawing of the patient whose cord is represented in Figure 160, shows the wasting and flaccidity of the paralyzed muscles. Figure 155 shows a child suffering from acute anterior poliomyelitis (infantile paralysis) affecting the lumbosacral enlargement. The *child is struggling,* yet the legs hang flaccid and paralytic; their lower motor neurons are destroyed by infection. Figure 154 shows wasting of the right arm caused by acute poliomyelitis in infancy, and Figures 158 and 159 show wasting of the abdominal muscles and of the glutei, from the same cause. Figure 164 shows the destruction of anterior column cells by central gliosis or syringo-myelia, and 163 shows the consequent paralysis and wasting. Figures 151, 152 and 165 show lower motor neuron paralysis in different regions due to disease or injury of

peripheral nerves. Figures 174, 175, 178 and 179 show cases in which softening of the brain stem has involved emerging roots of cranial motor nerves and has caused lower motor neuron paralyses of the muscles supplied by these, mixed with symptoms due to other tracts involved in the lesions.

PHYSIOLOGY OF THE UPPER MOTOR NEURONS

The cell bodies of the upper motor neurons are the large pyramidal (Betz) cells in the anterior central convolution (Fig. 27). The extent of the distribution of these cells is seen in Figures 90, 91, 93, 140, 141, 142 and 143. It is possible that smaller pyramidal cells take the place of the Betz cells in the lower third (face area) of this convolution. While there is considerable variation and overlapping in cerebral localization, the general arrangement is indicated in Figures 90 and 93. Broadly, the larynx, tongue, head and face area occupies the lower two-fifths of the anterior central gyrus, the arm area occupies the middle two-fifths, and the trunk and leg area occupies the upper fifth. The portion of the anterior central convolution on the medial surface of the hemisphere represents the perineal muscles (Fig. 93). The dendrites of these cells are in intimate contact with afferent axons from the optic thalamus and from almost all parts of the cerebral cortex (Fig. 27).

The axons of these cortical motor cells form the pyramidal tract. They converge toward the internal capsule, where they form the knee and anterior two-thirds of its posterior limb (lenticulo-thalamic portion), (Fig. 87, b, c, e). There is considerable mixing of the fibers, but the knee of the internal capsule is occupied almost exclusively by fibers controlling cranial nerve nuclei, and in the posterior limb arm and leg fibers succeed each other in this order from before backward (Fig. 87, b, c, e). In the mesencephalon (Fig. L; compare Figs. 87, d and 79, c) the pyramidal fibers occupy the middle three-fifths of the basilar portion; here the cranial nerve fibers are medial, the arm fibers next, and the leg fibers outside (Fig. L); there is, however, more mixing of the fibers in the mesencephalon than in the internal capsule. As the pyramidal tract enters the mesencephalon, a few small bundles split off (Dejerine's aberrant pyramidal fibers) and lie beside the lateral edge of the substantia nigra (Fig. 79, c). In the upper part of the pons these have reached the medial edge of the medial lemniscus (Figs. 79, e and 64) where they are easily distinguished as small, rounded bundles of fibers smaller than those of the lemniscus, and in fœtal specimens myelinating later. These are destined for cranial nerve nuclei both homolateral and crossed. There are

also fibers for the cranial nerves among the medial ventral border of the basis pedunculi (Fig. 79, c). In the mesencephalon, pyramidal fibers are also given off to the opposite nuclei of the third nerve. The nucleus of the fourth nerve is probably controlled from the third nucleus by way of the medial longitudinal bundle.

As the pyramidal tract enters the pons it breaks up into bundles (Figs. J, 65 and 17), but becomes concentrated again as it approaches the medulla oblongata (Figs. H, 16 and 63) where it forms a compact tract. In sections of an adult pons the pyramidal fibers are blended in the various bundles with the fronto-pontine and temporo-pontine fibers so that they cannot be differentiated, but in fœtal specimens at term the pyramidal fibers are partly myelinated, while the fronto-pontine and temporo-pontine fibers are not myelinated at all. Pyramidal fibers in the pons show little division into head, arm, and leg fibers. They give collaterals to pontine nuclei, and near the upper border of the pons supply fibers, chiefly crossed, to both fifth nuclei. At the lower end of the pons they give fibers to the opposite sixth nucleus, and to the seventh nuclei. For the upper face muscles they supply the seventh nuclei of both sides, but chiefly that of the opposite side; while for the lower face muscles they supply fibers to the opposite nucleus only.

When the pyramidal tract leaves the pons it forms the compact pyramid of the medulla oblongata (Figs. G and 60). Here also are one or two small aberrant bundles, chiefly in the medial lemniscus. Some of these are destined for homolateral distribution to the nucleus ambiguus, the lower nucleus of the eleventh nerve, and the nucleus of the first cervical (these last supply the muscles which turn the head in coördination with eye-turning movements), and to the twelfth nucleus. Heterolateral fibers are also given off here to all these nuclei on the opposite side. Indeed it will be observed that while those paired muscles which habitually act together have a bilateral pyramidal supply, the supply to the muscles of the opposite side is always in preponderance. In the case of the arm and leg it is probable that the homolateral pyramidal fibers are mainly distributed to the muscles controlling the roots of the limbs, while the more distal limb segments depend more completely on the opposite cerebral hemisphere.

At the lower end of the medulla oblongata (Figs. E, 58 and 79, j) approximately three-fourths of the pyramidal tract crosses in the pyramidal decussation to form the lateral cerebrospinal tract (crossed pyramidal tract), the remaining fourth forming the ventral cerebrospinal tract (direct

pyramidal). The fibers of this latter tract cross gradually in the white commissure, the decussation of the cord. The direct pyramidal tract is usually described as becoming exhausted about the mid-thoracic region, but it may often be traced as low as the lumbar enlargement (Fig. 79, n). On the other hand in a considerable number of cases the crossing is complete at the pyramidal decussation in the oblongata, or may be complete on one side and incomplete on the other. A ventral cerebrospinal tract is absent in most animals, but is fairly constant in man and higher apes.

In the first cervical segment of the cord, just after the pyramidal crossing, the *lateral cerebrospinal tract* consists of somewhat scattered bundles separated by neuroglia and nonmyelinated fibers. It forms the vertical fibers of the formatio reticularis of the spinal cord (Figs. 1, 56, and 57). Soon it becomes concentrated. It is at first separated from the surface by the dorsal spinocerebellar tract, but in the lowest dorsal and lumbosacral region it reaches the surface of the cord (compare Figs. C and A; also 55, 54, 53 and 79, k, l). It can be traced to the last sacral segment of the cord.

Homolateral pyramidal fibers. Besides certain aberrant strands of pyramidal fibers in the mesencephalon, pons, and medulla oblongata, which are largely destined for the supply of the homolateral motor nuclei of the fifth, seventh (for upper face muscles only), ninth, tenth, eleventh, and twelfth cranial nerves, a few fibers descend in the lateral cerebrospinal tract of the side from which they sprang, and are distributed to trunk muscles which always act in concert on both sides of the body. Such are the intercostal and abdominal muscles. This homolateral supply, however, is always comparatively feeble and subserves gross movements, not specialized movements (Fig. 79, k to n).

Mode of ending of the pyramidal fibers. The pyramidal fibers by collaterals and terminals carry voluntary motor stimuli from the motor area of the cerebral cortex to the dendrites of the lower motor neurons. These pyramidal fibers represent coördinated purposive movements rather than contractions of individual muscles, and each fiber is connected with many lower motor neurons. The pyramidal fibers, however, end in the cord segment by segment and there is evidence that according to the general rule the fibers that have to travel farthest down the cord are on the surface. The balance of evidence is against a direct synapse. Nowhere are degenerated pyramidal fibers seen to end in intimate relation with the lower motor neuron cells in the cranial nerve neuclei or in the anterior gray column of the cord. There appears to be an intercalated cell between

each upper motor neuron fiber and the lower motor neuron cells or, more probably, a group of these. This hypothesis that the fibers end round a group of intercalated cells seems better to explain the great excess of lower motor neurons over pyramidal fibers, and would account also for the representation of a vast number of combinations of movements in a comparatively small area of the cerebral cortex.

Function of the upper motor neurons. The pyramidal system appears last in order of development and is immeasurably more perfect and more dominant in man than in animals, even in the highest apes. In dogs its dominance is very slight compared with that in man. Conversely, there is no animal so dependent on its pyramidal system as is man. In man it is the only tract by which voluntary control can be exerted over the lower motor neuron and thus over striped muscles; but it is to be remembered that the pyramidal tract secures voluntary coördinated movements by using the whole subcortical reflex mechanism, whether it be controlled by the spinal cord, cerebellum, or the basal ganglia. Destruction of a pyramidal neuron in any part of its course removes to that extent an activating or controlling influence from the whole subpyramidal (infracortical) motor mechanism, or as much of it as lies below the lesion.

The contrast between the effects of a lesion of the upper motor neuron and one of the lower can be better realized after a study of Figure 100. A pure pyramidal lesion removes only cortical control, leaving all the other controls shown in the figure intact. Most capsular lesions remove the pyramidal and also the pallidal (lentiform) control but leave intact the cerebellar, vestibular, and other minor controls. A complete cord lesion removes all controlling and tonic brain influences. On the other hand a lower motor neuron lesion cuts off from the muscles all nervous influences whatsoever. By far the most marked result of a pyramidal lesion is loss of voluntary motion. The extent of this loss is in direct proportion to the extent of the lesion. For example, blocking of the vessel to the arm area in the cortex, or destruction of this area by a tumor means either weakness or else complete paralysis of the opposite arm according to the relative completeness of the destruction of the nerve cells. The same result would be brought about by destruction of the axons anywhere below the cell bodies.

In cortical and capsular pyramidal lesions some muscles of the opposite side of the body usually escape entirely or become weak, not paralyzed.

In a capsular hemorrhage, after the initial shock wears off, the eye muscles usually recover probably because lower centers such as the

superior colliculi or cerebellum assume control, under the influence of the calcarine area. Also the upper face muscles, the tongue, larynx, and pharynx appear on superficial examination to have escaped, but more careful study shows that they are distinctly weakened. Probably under the influence of their homolateral pyramidal supply they show syncinetic movements when vigorous voluntary efforts against resistance are made with the muscles of the well side. This is also true of the muscles for head-turning as well as the trunk, intercostal and abdominal muscles, and perhaps those of the shoulder and pelvic girdles.

Horsley removed the arm area of the anterior central convolution for the relief of athetosis. He found that his patient ultimately acquired some control over the forearm and hand muscles. Horsley thought that this was probably due to an indirect motor control from the posterior central convolution by way of the cerebellum through the red nucleus. The basal ganglia might also come in, but no adequate explanation suggests itself to the writer. In dogs, complete removal of the motor cortex of one side produces only very temporary paralysis; the cerebellum and basal ganglia being capable of performing stock movements. This does not apply to man except to a very limited extent, for of all animals man has the highest development of the cerebral cortex and is most dependent upon its activities and dogs lose highly specialized movements such as giving a paw.

Besides paralysis a pure pyramidal lesion usually carries with it a *loss of cutaneous reflexes* for the part involved, and an increase of tendon reflexes, and, if the leg area or fibers be involved, the Babinski phenomenon. The reason for the loss of cutaneous reflexes is not known. The increased tendon reflexes would seem to indicate that the upper motor neurons exert a controlling influence over spinal reflexes. The *Babinski phenomenon*, also called the extensor plantar response, consists of reflex extension of the great toe and fanning of all the toes when the outer margin of the sole of the foot is stroked with a match or with the finger nail. The normal reflex response to scratching the sole of the foot in this manner is flexion of the toes, but in infants before the pyramidal tract is fully developed, the response is normally an extension of the great toe. When there is more or less destruction of pyramidal fibers to the leg segments of the spinal cord the Babinski phenomenon or extensor plantar response takes the place of the normal flexion. This sign is absent, however, in certain toxic conditions and when there is any interruption of the reflex arc from the sole of the foot. The clinician must remember

that the Babinski phenomenon alone is not proof of an organic pyramidal lesion; it has been observed in the toxic states enumerated below, viz.:

(1) After epileptiform fits.

(2) In severe hepatic insufficiency either in acute cholemia or in acute exacerbations of chronic cholemia, with or without jaundice.

(3) In the very deep sleep of exhaustion.

(4) After puerperal eclampsia.

(5) In hypoglycemic coma from an overdose of insulin.

(6) In acute uremia after epileptiform convulsions.

(7) In lethargic encephalitis.

(8) In poisoning by certain drugs: in delayed chloroform poisoning; in coal-gas poisoning; in poisoning by sulphonal and veronal, and by scopolamin. The last of these drugs has been used for diagnostic purposes; 1/150 grain scopolamin may emphasize a doubtful Babinski reaction.

The occurrence of the extensor plantar response in these conditions is interpreted to mean that the toxicity of the blood causes temporary derangement of the cells or fibers of the pyramidal pathway. The influence of the pyramidal tract on the whole tonic mechanism is largely inhibitory or at least modifying in its nature. It follows that a pure pyramidal lesion removes at least one control over muscle tone and a certain amount of muscular rigidity accompanies the paralysis. The rigidity is not so marked, however, as it is in a capsular lesion, when the lentiform control also is removed.

If the pyramidal lesion be above the pyramidal decussation the resulting paralysis is on the opposite side of the body. If the lesion be below the decussation, as for example if the cord be cut halfway across by a stab wound, the paralysis is on the same side as the lesion. A lesion of both pyramidal tracts in the cord as by total transverse or double pyramidal lesion causes paralysis of both lower limbs (paraplegia).

From its anatomical relationships few lesions of the pyramidal system are limited to this system. Thus a hemorrhage into the internal capsule, the ordinary form of apoplexy, also destroys more or less the lentiform nucleus or its efferent fibers (pallidorubral and other fibers). It produces, therefore, symptoms due to the combined loss of pyramidal and of pallidal control of the lower motor neurons. Also the pyramidal fibers to the whole of the opposite side of the body are involved to a certain extent. The result is that such a hemorrhage into the internal capsule causes loss of voluntary control over the opposite side of the body,

increased reflexes, and the Babinski phenomenon, all due to the pyramidal lesion; it also causes rigidity of the paralyzed muscles which is partly due to the loss of pyramidal control, but is mainly due to the loss of pallidal control. In such a lesion, muscles with bilateral pyramidal supply are only partially affected, weakened but not paralyzed, and because of the action of lower reflex centers, the eyes soon regain their movements. The symptoms of pyramidal lesions vary greatly according to the exact location of the lesion and the extent to which neighboring tracts are implicated; but enough has been said to explain the neurological principles underlying the symptomatology.

Examples of Upper Motor Neuron Lesions. If the anatomical limits of the lesion are known, the symptoms of a pyramidal lesion can be worked out with accuracy, just as, conversely, in a living patient, one can determine the location of the lesion from the symptoms. Figures 79, a and b, illustrate a case of cortical softening produced by occlusion of the middle cerebral artery. A softening of this extent and position would give rise to aphasia, if the patient be right-handed, and probably paralysis of the right arm. As seen in 79b the deeper softening would probably involve leg fibers also so that the result would be paralysis of the right face, arm and leg (right hemiplegia). Figure 168a shows an old softening or hemorrhage in the right internal capsule, and Figure 196 shows a patient who has partly recovered from such a lesion but still has a spastic paralysis of the left side of the body. Figures 194 to 199 show various attitudes assumed by the rigid limbs in upper motor neuron paralysis. Figure 171 shows lesions affecting both pyramidal tracts in the cord causing spastic paralysis of both legs, and Figure 206 shows the rigid contracted paralyzed limbs of such a case. Figures 172 and 205 illustrate another variety of spastic paraplegia. Figure 170, a to e, shows slow destruction of both lateral cerebrospinal tracts in a case of chronic syphilitic disease of the cord and Figures 204 and 206 show patients suffering from the form of paralysis produced by such a lesion. The figures 202 to 207 show typical paraplegic attitudes. Figures 174, 175, 178, 180, 182 and 183 show typical lesions of the brain stem involving the pyramidal tract as well as other neighboring structures.

Degenerations. Whenever the pyramidal tract is interrupted there is rapid degeneration of the fibers of the tract below the lesion, owing to their separation from the parent cells. There is also chromotolysis and gradual disappearance of the upper part of the neurons which have ceased to functionate, but this is a much slower process. There is no degenera-

tion of the lower motor neurons or of the muscles (see Figures 79; 168, a and b; 169, a to f).

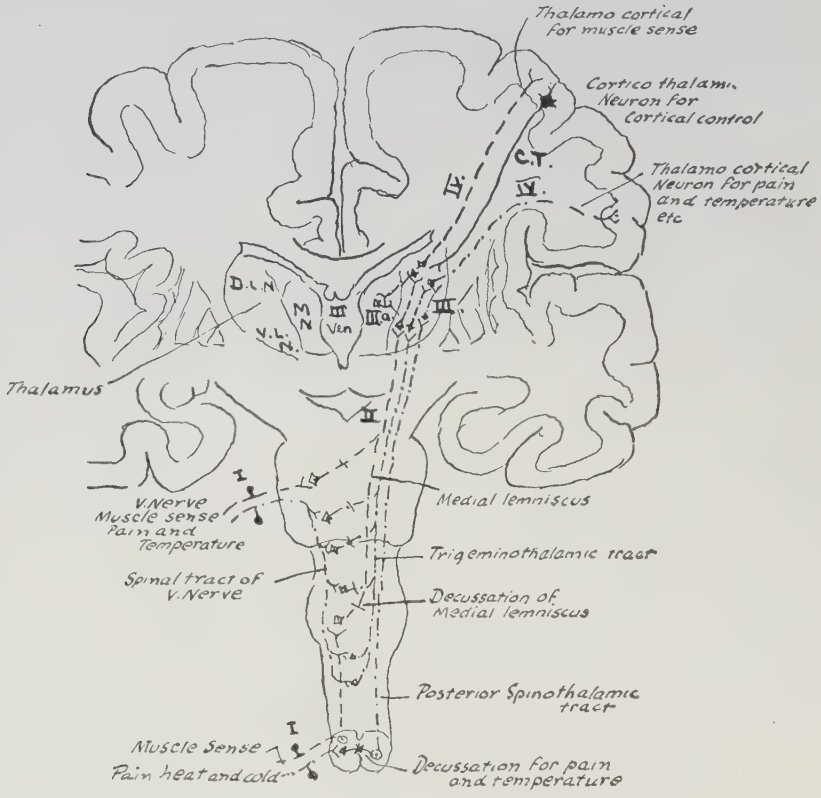


Diagram of the four sensory neurons.

THE SENSORY NEURONS (see figure above)

The sensory neuron system is much more complex than is the motor neuron system. In its simplest form it consists of at least four neurons. These are:

(1) The lowest sensory neuron (L.S.N.).

(2) The second sensory neuron, relaying the lowest sensory neuron (L.S.N.) to the ventrolateral nucleus of the thalamus.

(3) The third sensory neuron, relaying 2 to the dorsal part of the lateral thalamic nucleus.

(3a) A variant of 3, consisting of a relay from the terminal nuclei of either the second or the third sensory neuron to the essential thalamic organ (Head and Holmes).

(4) The fourth sensory neuron, passing from the dorsolateral thalamic nucleus, or from the essential thalamic organ to the posterior central convolution and perhaps to other areas of the cerebral cortex.

All these relays are necessary for the full representation of ordinary sensory impulses in consciousness.

In addition to this certain of the lower sensory neurons convey impressions of muscle sense for transmission to the *cerebellar coördinating and tonic mechanism.* These sensations never appear in consciousness. They involve:

(1) Certain lowest sensory neurons;

(2) A relay from these directly to the cerebellar cortex, mainly that of the vermis, or to the vestibular nuclei and thence to the vermis; or to the inferior olive and thence to the cortex of the cerebellar hemisphere. From the cortical (Purkinje) cells of the hemispheres commence the cerebellar efferent tracts to be taken up later.

The sensory system is still further complicated by the special senses, smell, taste, sight, hearing, vestibular sense, and visceral sense, each of which has its special mechanism.

Further, as we have seen, conscious sensation of the ordinary type is itself very complex. It consists of *three types, epicritic, protopathic and deep sense.* A detailed description of these will be found on page 142, but a brief summary will be of value here.

Epicritic sense or tactile sense proper includes light touch, tactile localization; skin pain localization; tactile discrimination; heat and cold between 26° and 37° C. (a little below and up to or a little above blood heat).

Protopathic sense includes the perception of skin pain, as from pin prick or pinching; the sensation of heat over 38° C., and of cold under 24° C.; the sense of touch as conveyed by the hairs, and of tickling.

Deep sense includes pressure sense; pressure localization and pressure pain; vibratory sense; and muscle-tendon-joint sense, including the sense of passive motion and of passive position, the sense of weight, and stereognostic sense.

All these forms of sensation are grouped together in the same posterior spinal nerve roots, but they are differently distributed in the sensory paths of the spinal cord and brain stem (contrast Fig. 99 with Fig. 69).

The lowest sensory neurons for pain and temperature sense and for hair sense are probably represented by the smaller cells of the posterior root ganglia. The single axons of these cells are unmyelinated, and each

splits into a peripheral and a central process (Fig. 43). Ranson has demonstrated that in cats these unmyelinated fibers convey pain and he believes that they also convey the more marked degrees of heat and cold. On theoretical grounds the author would add to this hair sense. In fact these unmyelinated lowest sensory neurons are probably the neurons for all protopathic sense. Their peripheral processes end in some form of sensory end organ in the skin (Figs. 45, 46, and 49); their central processes enter the posterolateral column (Fig 69) as unmyelinated fibers, and divide each into an ascending and a descending branch. The ascending process runs up in this column 2 to 6 cm. according to the region of the cord. The axons send collaterals into the posterior gray columns and are probably relayed by the neurons whose cells are scattered in the gelatinous substance and whose axons cross in the gray matter and gray commissure, at a level not more than 2 to 6 cm. above the point of entry of the original nerve root into the cord. Together they form the **posterior spinothalamic tract** whose location is indicated in Figure 69, A, B, and C; and 81, a to g.

Here different fibers convey pain, heat and cold, but all are intermingled; in this one tract all grades of heat and all forms of pain are conveyed. This statement may be taken to cover the vast majority of cases, though it is possible that exceptions occur. (Thus Dejerine quotes a case which seems to show that in some cases at least pressure pain is conveyed homolaterally by the posterior column.) In the cord **the posterior spinothalamic tract** is medial to the ventral spinocerebellar tract and ventral to the rubrospinal tract and is more or less mixed with both of these (Fig. 69). In the medulla oblongata it is medial to the gelatinous substance (Figs. F and G), a position which it holds in the lower pons (Fig. H). In the upper pons it is dorsal to the junction between the medial and lateral lemnisci (Fig. J), and above this it blends with the medial lemniscus. With the medial lemniscus it ends in the ventrolateral nucleus of the thalamus (M.).

It should be emphasized that the crossing is in the cord shortly above the entry of the nerve roots, and that while different peripheral nerves carry different varieties of pain and grades of temperature, all grades of temperature and all varieties of pain are grouped together in the same tract in the cord and brain stem.

Pain and temperature neurons of the fifth cranial nerve have cell bodies in the semilunar ganglion and peripheral processes ending in special sense organs for pain and temperature distributed throughout the whole cutaneous and mucous area supplied by this nerve. This area comprises

the face, the anterior half of the scalp, the conjunctiva, the eyeball, the nasal mucosa, most of the mouth and the anterior two-thirds of the tongue. The central processes of these neurons pass by way of the sensory root of the fifth nerve into the tegmental region of the pons (Fig. I). Here they enter the spinal tract of the fifth nerve and, descending in it, arborize in the gelatinous substance. The spinal tract of the fifth and the associated gelatinous substance are homologous with the posterolateral column and gelatinous substance of the cord, with which they are continuous. Like the posterolateral column the spinal tract of the fifth nerve is very rich in unmyelinated fibers. It can be traced by degeneration methods downward into the upper cervical segments of the cord, where it blends with the sensory roots of the upper cervical nerves. Clinical cases prove adequately that the spinal tract of the fifth nerve conveys sensations of heat, cold, and pain. It is relayed by arcuate fibers to a distinct tract on the opposite side of the medulla oblongata called the **trigeminothalamic tract** (Figs. F to J). Clinical evidence also proves that this tract is distinct from the posterior spinothelamic tract, lying in the formatio reticularis nearer the median plane. The trigeminothalamic tract joins the medial lemniscus in the mesencephalon or hypothalamic region, and so enters the ventrolateral nucleus of the thalamus.

Glossopharyngeal fibers for pain, heat and cold from the posterior third of the tongue probably join the spinal tract of the fifth nerve.

The **vagus** contains a few fibers conveying pain, heat and cold and touch from parts of the pinna, concha, and back of the ear. This branch of the vagus is called the auricular branch. The **facial nerve** contains, in some cases, similar sensory fibers of about the same distribution. These are inconstant and are probably vestigial in character. The writer believes that the fibers of both these nerves subserving the senses of pain, heat, and cold, join the spinal tract of the fifth nerve and have similar central connections.

Sensory neurons for simple touch. The lowest sensory neurons for simple touch have cell bodies in the posterior spinal root ganglia. The central processes of these are probably the fine myelinated fibers of the posterolateral column. The peripheral processes end in some form of touch corpuscle. The central processes enter the cord with the other fibers of the posterior nerve roots, and after that the path for touch appears to vary in different individuals. Clinical cases seem to indicate that there is a path for touch in the homolateral posterior column, relayed in the nucleus gracilis for fibers below the midthoracic region of the cord,

and in the nucleus cuneatus for higher segments. However, in perhaps half the cases observed there appears to be a relay in posterior column cells 2 to 4 segments above the level of entry. In these cases the axons of the new relay cross in the white or gray commissure and form the *ventral spinothalamic tract* (Figs. 69, A, B, and C), and thus ascend in the side of the cord opposite to entering nerve roots, and join the lateral lemniscus in or near the mesencephalon. This ventral spinothalamic tract conveys touch and pressure sense for the opposite side of the body (Page May). In a small percentage of cases the crossing is complete. The central tracts for touch and pressure for the trigeminal nerve are unknown.

The spinal paths for muscle-tendon-joint sense are all in the homolateral posterior column. These paths convey the sense of passive motion, weight as estimated by muscular action, tactile discrimination, stereognostic sense, and tuning-fork vibration, but do not convey muscle and joint pain. From the posterior column all are relayed in the *nucleus gracilis or cuneatis,* and from these nuclei all ascend to the thalamus in the *opposite medial lemniscus* with the exception of tactile discrimination, which follows an independent path in the medulla oblongata and pons. We shall therefore follow this group of sensory paths together.

This group of lowest sensory neurons are all myelinated. Their cells belong to the large-celled groups of the posterior spinal ganglia (Fig. 43). Their peripheral processes end in various types of end organs in skin, muscles, joint capsules or periosteum (Fig. 48). Their central processes enter the lateral peripheral area of the posterior white column (Figs. 69, and A, B, C) and split each into an ascending and a descending branch (Fig. 74); each branch gives many collaterals to the spinal gray columns for purposes of spinal reflexes. The descending branches pass through many segments of the spinal cord, so that branches from the sensory nerves of the cervical plexus can be found in the sacral segments (Fig. 81, q to w). Thus coördinating or reflex paths are provided between the upper and lower limbs. These descending branches at first suffer very rapid diminution as they run downward; later they diminish much more slowly (Figs. 80, g to j). There is a tendency for the longer roots to shift medialward and dorsalward. Because of their change of location at different levels the descending tracts of the spinal nerve roots are variously named—*fasciculus interfascicularis* (comma tract), *Flechsig's oval field, septomarginal bundle of Bruce, sacral triangle of Philippe and Gombault, bandelette of Hoche,* and *cornucommisural tract* (Figs. 81, r to w). All these are composed solely of descending root bundles, except

the cornucommissural tract of Dufour, which may be partly composed of endogenous association fibers (Fig. 78, Batten and Holmes, *Brain,* 1913). The ascending root fibers, like the descending branches, send collaterals into the gray matter and they also diminish at first rapidly, then more slowly as they pass upward. The long fibers shift medially and dorsally as they ascend, so that long fibers from the leg and the lower part of the trunk form the *funiculus gracilis* (Fig. 80), and long fibers from the upper part of the trunk and from the arm occupy the dorsal part of the *funiculus cuneatus* (Figs. A to E). It has previously been noted that the long ascending fibers myelinate somewhat later than the short fibers and are finer in caliber. The short fibers provide for spinal reflexes and coördination; the long fibers are the conducting paths to the thalamus. These long fibers reach the *nucleus gracilis* and the *nucleus cuneatus* and are here relayed. The cells of these nuclei are the *second sensory neurons* and their axons form the *deep* or *internal arcuate fibers* (Figs. E and 59). These intercross in the raphe of the medulla oblongata, and each group forms the opposite *medial lemniscus* (Fig. 59). The ventral part of the medial lemniscus comes from the nucleus cuneatus (Figs. 51 and 52), the dorsal part from the nucleus gracilis. The *medial lemniscus* thus becomes the tract for the muscle-tendon-joint sense complex, vibratory sense, and, in some cases at least, simple touch and pressure sense from the opposite side of the body. Concerning the path for tactile discrimination, there is clinical evidence to indicate that it is here segregated to a special tract in the formatio reticularis (Head). In the mesencephalon or subthalamic region all tracts for ordinary body sense blend in the *medial lemniscus* (Fig. J), and all these end in the ventrolateral nucleus of the thalamus (Fig. M). From a clinical standpoint the fact of chief importance is that the sensory paths for pain, heat and cold decussate in the cord shortly after the entry of their respective nerves, whereas the paths for all other forms of body sense ascend in the posterior columns homolaterally to the level of the medulla oblongata before crossing to join the opposite medial lemniscus. However, in a small percentage of clinical cases the path for touch and pressure crosses completely in the cord.

The paths for touch, pressure and muscle sense for the fifth nerve. The spinal tract of the fifth nerve and the associated gelatinous substance are serially homologous with the posterolateral tract and the gelatinous substance of the cord, and their function is the conduction of pain, heat and cold. Following this comparison one cannot but see a close resemblance between the structure of the *lateral sensory nucleus of the fifth*

nerve and that of the nucleus gracilis or the nucleus cuneatus. This resemblance leads the writer to believe that the sensory fibers of the fifth nerve which convey muscle sense from the muscles of mastication and probably from most of the tongue muscles end in the lateral nucleus (Fig. I). This nucleus is probably relayed in the opposite medial lemniscus to the thalamus (Fig. I and 63). When the semilunar ganglion of the fifth nerve is removed surgically for the relief of neuralgia, pain, heat and cold are lost over the area of distribution of the nerve, but, in some cases at least, pressure sense is still conducted. Spiller believes that this conduction of pressure sense is subserved by the facial nerve. If this be so, and in the cases quoted it can scarcely be otherwise, it is probable that those facial fibers conducting pressure sense end in the lateral sensory nucleus of the fifth nerve. Muscle sense for the tongue and face muscles is probably a function of the fifth nerve and is conveyed by fibers which end in the trigeminal lateral sensory nucleus. No definite data are known to the writer, but it is to be presumed that tactile discrimination and tuning-fork vibration for the face and tongue are also relayed in the lateral sensory nucleus of the fifth nerve.

Sherrington has shown that section of the ophthalmic divisions of the fifth nerve does not affect the action of the muscles which control the eyes, and he argues that the third, fourth, and sixth nerves contain fibers which convey muscle sense for the eye muscles. The ganglionic cells, end nuclei, and central connections of the sensory nerves for the eye muscles are still unknown.

Grouping of the second sensory neurons in the mesencephalon and subthalamic region. All forms of bodily sensation are relayed by second sensory neurons to the thalamus. Though separated in the cord and brain stem, these tracts lie close together in the mesencephalon and subthalamic region, so that although they are often involved separately in lesions lower down, they are seldom involved separately in the mesencephalon, subthalamic region, or ventral nucleus of the thalamus. Thus a complete lesion of the medial lemniscus in or above the mesencephalon causes the loss of all ordinary forms of sensation for the opposite side of the body, including the head.

Third sensory neuron (see figure, p. 134). From the ventral nucleus of the thalamus two separate relays occur. One of these is by short neurons to the *essential organ of the thalamus* (Gordon Holmes). This essential thalamic organ is postulated on clinical evidence, but has not been demonstrated anatomically. Its exact location is not settled, but it

is probably the medial nucleus. In this essential thalamic organ most forms of sensation, certainly heat, cold, pain, and the sense of roughness or smoothness of texture, reach consciousness. They are here chiefly associated with feelings of comfort or discomfort, pleasure or pain, and probably also with emotional responses of a reflex character such as involuntary facial expressions of pleasure, pain, disgust, or anger. Goltz (Schaefer's Physiology) removed from a dog the whole of both cerebral hemispheres, injuring slightly also the corpora striata and the thalamus. The dog reacted to discomfort by snarling and barking, to loud sounds by shaking the ears. If a foot were placed in cold water, it was withdrawn. Meat made bitter with quinine was rejected. These reflexes imply an efferent path from the thalamus which does not go by way of the cortex, but which is probably quickly relayed by a longer descending tract from some such nucleus as the globus pallidus, the red nucleus or the corpus hypothalamicus.

There are also short internuclear neurons from the ventrolateral to the dorsolateral thalamic nucleus. The various forms of bodily sense are very much concentrated in the ventral portion of the lateral thalamic nucleus. Clinical observations seem to indicate a wider distribution and regrouping of these in the dorsal part of the lateral thalamic nucleus.

Fourth sensory or thalamocortical neuron. The thalamus is connected with the cortex, first by a thalamocortical relay by which impressions of temperature, pain, etc., received by the thalamus may be recorded and compared with previous experiences and thus made into a groundwork for cortical activities. This connection occurs somewhere in the parietal region of the cortex. Secondly there is a corticothalamic connection from cortical regions unknown but probably extensive and various by which a cortical control can be exerted over thalamic reactions. Fig. M. Much of the intricate detail of the thalamic mechanism is still theoretical; it is founded on clinical symptoms of limited thalamic lesions (Head and Holmes, *Lancet,* 1912). See thalamic lesions, page 214.

PHYSIOLOGY OF THE SENSORY NERVES AND PATHS

The posterior root of a spinal nerve carries all forms of ordinary sensation to the spinal cord (Figs. 69 and 99). On account of the extreme importance of differentiating these forms of sensation, it is necessary for the student to have a vivid conception of the methods of testing the different types. Many mistakes are made even by experienced practitioners, and some supposed peculiarities noted in individual cases are

the result of a confusion of epicritic heat and protopathic heat, hair sense and tactile sense, etc. This confusion offers the excuse for again summarizing the types of ordinary body sense and the tests for them.

Epicritic Sense:

(*a*) Tactile sense proper is tested by a delicate fluff of absorbent cotton on hairless skin.

(*b*) Heat and cold between 26° C. (78° F.) and 37° C. (99° F.). (Note 37° C. is normal body temperature, viz., 98.6° F.) It is tested by test tubes, preferably metallic, containing water at carefully graded temperatures, or by teaspoons in glasses of water of graded temperature.

(*c*) Touch, temperature and pain localization. These nerves do not convey pain as such, but their integrity is necessary for the distinct localization of touch, pain and temperature sensations, which otherwise have a diffused character.

(*d*) The discrimination of two blunt points of a compass as two when touching the skin simultaneously.

Protopathic Sense:

(*a*) Skin pain as tested by pin prick; hair pain caused by pulling hairs.

(*b*) Heat over 38° C. (100° F.) and cold under 24° C. (75° F.)

(*c*) Touch as conveyed by the hairs, and tickling as when hairy parts are brushed by a fluff of absorbent cotton.

In the absence of epicritic nerves these sensations are very much diffused, and the integrity of the epicritic nerves is necessary for accurate localization.

In the peripheral distribution of the nerves (Fig. 99), the cutaneous nerves convey both epicritic and protopathic sense; myelinated fibers convey epicritic sense, and nonmyelinated fibers convey protopathic sense. The conveyance of skin pain by the nonmyelinated fibers in cutaneous nerves has been proved by Ranson in cats. It is probable that marked degrees of heat and cold and hair sense are also conveyed by nonmyelinated nerves. Hence in testing epicritic sense extremes of temperature are to be avoided, and it is necessary to shave hairy parts for accurate determination of light touch perception. At least in the hands and feet, where sensation is highly developed, epicritic nerves do not overlap in their areas of distribution; protopathic and deep sensory nerves on the other hand overlap considerably.

After section of a nerve and subsequent suture the first fibers restored are protopathic (nonmyelinated) nerves, and thus protopathic sense is

restored long before epicritic sense, but the localization of pain and touch is very imperfect until the epicritic nerves are regenerated.*

Deep Sensibility. This includes:

(*a*) The sense of pressure and pressure localization. Touch as tested by a pinhead, pencil point, or even too stiff a pencil of absorbent cotton is included in this form of sensation and not in epicritic sense.

(*b*) Pressure pain, as produced by deep pressure with a pencil; also joint pain, and muscle pain.

(*c*) Muscle-tendon-joint sense, as indicated by the sense of movement and of the position of the limbs, fingers, or toes, the sense of weight, and stereognostic sense.

(*d*) Vibratory or tuning-fork sense as tested by a large tuning fork. This is often spoken of as bone sense, but it is also conveyed by muscles as is easily demonstrated on the abdominal wall.

Deep sensibility is conveyed by afferent nerves which run with the muscular nerves and with the afferent nerves of tendons. Thus, even if all the cutaneous nerves to the fingers be severed, the fingers are still sensitive to deep pressure unless the tendons also be cut.

Distribution of Peripheral Sensory Nerves. Clinically one has often to distinguish between nerve-root distribution and the distribution of peripheral nerves. From the third to the eleventh thoracic nerves, each nerve root entering a corresponding cord segment carries sensation from a nearly horizontal area of the skin of the trunk (Fig. 96, a, b and c). This does not correspond with the skin immediately over the intercostal space to which the nerve belongs. There is, however, considerable overlapping of neighboring nerves so that in the trunk there is no marked loss of sensation until three successive nerves are divided. It is doubtful whether this overlapping in the trunk is true for man (see *Revue Neurologique,* December, 1926).

Reference to Figure 96 shows that, in the limbs, the posterior spinal nerve roots as they enter the corresponding cord segments are represented by longitudinal strips of skin, not by circular bands. This is not so difficult to understand if one thinks of the mode of development of the limbs. It is evident from the formation of the brachial plexus that the upper extremity is an outgrowth from those segments of the trunk that form this plexus, that is, it is an outgrowth from the body segments which correspond to the fourth to the eighth cervical and first and second thoracic

* But on this read Stopford "A New Conception of the Elements of Sensation," Brain, 1922-23.

segments of the spinal cord. As the bud of the forelimb grows, the shoulder, radial border of the forearm, and thumb are directed cephalward, the little finger, ulnar border of the forearm, and axillary border of the arm are directed caudalward (Fig. 101). One can imagine that the growing limb stretches the skin with its cutaneous nerves like a long glove. The first and last skin segments stretch but little; the fourth cervical and second thoracic spinal nerves are of short distribution and supply the shoulder and the axillary skin respectively (Fig. 96, a, b, c). The intermediate skin segments stretch farther so that the sixth, seventh, and eighth cervical spinal nerve roots come to supply strips down the back and front of the arm and forearm which reach the fingers. A comparison with Figure 101 shows a skin distribution for the cord segments quite closely corresponding to Figure 96, a, b, and c. The leg areas do not fit in so easily and are more difficult to remember, but one can usually refer to the figures for diagnostic purposes. Segmental, better called radicular, areas as mapped out by different neurologists do not quite agree, but the mapping is sufficiently accurate for clinical purposes. Comparing Figure 96, a, b, and c with 96, d, and e, or with corresponding figures in any textbook of anatomy, one finds that the skin pattern for the peripheral distribution of cutaneous nerves is quite different. The relation of sensory nerve roots to spinal cord segments is very obscure. The segmental distribution of sensation in the limbs does not correspond at all accurately with the radicular distribution of the corresponding nerves, especially in the upper limbs. In the forearms the segmental distribution of anæsthesia in case of a cord lesion tends toward a glovelike distribution. The cutaneous nerves on entering the cord appear to show a tendency to form their chief sensory relays in those cord segments that supply the chief muscles to the underlying parts of the skin area supplied. This distinction between segmental and peripheral nerve areas will be taken up more fully in dealing with lesions of the spinal cord.

Note. The segmental motor supply of skeletal muscles has also been worked out. Some of the chief points in segmental muscle supply are given in Figure 95. A full list of segmental muscle supply will be found on page 327. Usually each muscle is supplied by at least two cord segments.

Experimental section of posterior nerve roots. If at least three consecutive posterior nerve roots be sectioned proximal to the ganglia in the thoracic region, a corresponding area of skin on the same side of the trunk is rendered completely anæsthetic (Fig. 96, a and b). This area is a band, nearly horizontal, passing halfway round the body. Section of two consecutive nerve roots seriously blunts sensation, especially for pro-

topathic sense (pain, heat, and cold). In the limb regions in man there is less overlapping of segmental areas, and disease of two consecutive posterior nerve roots will give rise to a distinct longitudinal stripe of anæsthesia, especially anæsthesia to heat and cold. Experimental section, proximal to the ganglia of *all the posterior nerve roots* supplying a limb produces the following results:

1. Anæsthesia of the area supplied for all forms of sensation.
2. Ataxia of the corresponding limb.
3. Loss of reflexes, with loss of muscle tone and marked flaccidity.
4. Functional, though not actual paralysis of muscles.
5. Trophic lesions of the skin.
6. Degeneration in the posterior columns of the cord; chromatolysis in more than half the anterior gray column cells, and in all the cells of the nucleus dorsalis for the extent of the nerve section.
7. Atrophy of the corresponding muscles but not the reaction of degeneration (A. Rendle Short).

In spite of the extent of the nervous lesion there is usually no shock.

Let us discuss these results separately.

The *anæsthesia* needs no explanation. The anæsthetic area will be bordered by a narrow zone where sensation is defective but not lost, because of the overlapping of the supply from the uninjured nerve roots entering cord segments immediately adjacent.

Ataxia. By ataxia is meant difficulty in using muscles which are not paralyzed but which do not respond properly to voluntary calls. In order that we may control the use of our muscles we must be in constant touch with them by a flow of entering sensory impulses from the muscles themselves. If the sensory nerves from muscles be injured the patient does not know he has muscles and consequently cannot use them. For their coördinated use there must be also a constant flow of sensory impulses to the cerebellum. Besides this, part of the ataxia may be due to loss of the collaterals from the sensory nerve roots to the ventral gray column cells, which will interfere with the reflex mechanism of the cord itself.

Loss of muscle tone. This loss of sensory inflow from muscles also accounts for the loss of muscle tone and loss of reflexes. The arc for spinal reflexes consists of a peripheral sensory nerve, certain endogenous intercalated neurons, and the efferent cord neurons. If this arc be broken anywhere, cord reflexes are impossible.

Functional paralysis. After a monkey has had the posterior nerve roots for one arm cut, he never again voluntarily uses that arm. He does not know that he has it to use. It is not incapable of movement, however,

for he may possibly use it synchronously with the other arm, or to scratch reflexly a distant part of his body of which the sensory nerves are intact. Yet its movement is never a primary volitional act. Such loss of motion is functional as opposed to motor neuron paralysis.

Trophic lesions. The nutrition of the skin, the tone of its blood vessels, the regulation of the secretion of sweat all depend on a perpetual inflow of sensory skin impulses; any extensive sensory loss is always followed rapidly by defective skin circulation, and sometimes by skin eruptions, and intractable sores or local gangrene. With the lack of sensory impressions, reflex protection of the part from injury becomes impossible, and since pain, heat and cold are not appreciated, voluntary protection is greatly handicapped. The slight traumatism constantly being sustained adds greatly to the risk of ulcers, internal derangements of joints, and other similar lesions accompanying sensory paralysis, and it is difficult to differentiate between the effects of such constant mild injuries and true nutritional disturbances.

Degenerations (Fig 80). Within the cord the distal ends of the axons, being separated from the ganglia, undergo rapid Wallerian degeneration. This is most evident in the posterior columns above the lesions, and reaches as high as the nucleus gracilis or nucleus cuneateus, but the number of degenerated fibers rapidly diminishes. Marchi staining in acute lesions shows also degeneration of the descending branches of the entering nerve roots involved. As there are few myelinated fibers in the posterolateral column, this does not show degeneration by ordinary microscopic methods.

Chromatolysis. The cells of the nucleus dorsalis and of the anterior gray columns corresponding to those segments of which the posterior nerve roots have been cut, are largely deprived of afferent stimuli, cease to functionate and hence undergo chromatolysis. Later they may atrophy and disappear, so that ultimately there may be degeneration of their neuraxons.

Muscular atrophy. At first the muscles atrophy from lack of use and probably also from defective nutrition of a reflex character; but as the anterior gray column cells degenerate, the muscles may also undergo late electrical degenerative changes. Those anterior gray column cells which escape may supply muscles which habitually act with similar muscles of the opposite side of the body. They would thus be in touch with afferent impulses from uninjured nerve roots on the opposite side of the cord, and thus certain trunk muscles may escape atrophy and degeneration.

Tabes dorsalis is a widespread chronic affection of sensory nerve roots, and the effects of experimental section of these roots just described throw a flood of light on the symptomatology of this disease.

Posterior root ganglia. Intense neuralgia accompanied by a vesicular eruption are the chief symptoms of herpes zoster, which is now known to be due to an inflammation of one or more posterior root ganglia. The eruption is distributed over the skin area corresponding to the segment of the spinal cord with which the affected ganglion is connected. Intense neuralgic pain with more or less sensory loss and a herpetic eruption is characteristic of affections of sensory ganglia. The partial anæsthesia may be obscured by overlapping nerves. Many of the cases of trigeminal neuralgia may be due to disease of the semilunar ganglion of the fifth cranial nerve.

The spinal nerves distal to the root ganglia divide almost immediately into anterior and posterior rami (see Fig. 99).

Posterior rami of spinal nerves. The posterior rami of the spinal nerves are peculiar in that they form no plexuses and each has therefore a pure radicular distribution. With the exception of the great occipital they are not liable to injury separately. The posterior rami of the spinal nerves from the fifth cervical to the first thoracic, inclusive, do not reach skin areas. Similarly, the posterior rami of the fourth and fifth lumbar do not reach skin. The posterior rami of the sacral nerves do not call for individual differentiation. Thus the only posterior rami important to the neurologist are the second cervical and the second thoracic to the second or third lumbar, inclusive.

Anterior rami of the spinal nerves. The anterior rami of cervical nerves are peculiarly exposed to injury and may be taken to exemplify this phase of the subject. In Figure 99, if an anterior ramus be cut at the point B 2′, as by a stab wound in the neck, the results will be both motor and sensory, since here the roots have joined to form a mixed nerve.

The muscles supplied by this nerve will be paralyzed, but as most muscles are supplied by two segments the paralysis will be only partial unless more than one ramus is cut. The anæsthesia also may be slight unless two successive rami be cut, when the anæsthesia may be marked. The distribution will be radicular. Thus if the fourth, fifth and sixth cervical nerves be cut, there will be flaccid paralysis with rapid wasting and degeneration of the deltoid and biceps (Fig. 95) and anæsthesia of the skin of the shoulder and of the radial side of the arm and forearm

(Fig. 96, a and c). With so limited a lesion there would probably be sufficient afferent influences reaching neighboring segments of the cord to prevent local trophic lesions of the skin.

Incomplete lesions of peripheral nerves are usually associated with defective conduction with altered reaction to sensory stimuli producing sensation of disagreeable or even painful nature. There is not infrequently local pain, or pain referred to the peripheral skin areas represented by the cord segment involved. The paralysis may be more marked than are the sensory symptoms. Purves Stewart states as his experience in the late war that partial division of a nerve causes partial paralysis of the muscles supplied, together with burning pain referred to the peripheral distribution of the cutaneous branches. This burning pain (causalgia) is especially well marked in partial injuries of the median and tibial nerves; the burning pain affects the palm or sole as the case may be, and is often of great intensity. Hypertrophic changes in the skin, hair and nails and hyperkeratosis (excessive growth of epithelium) is occasionally met with. There may be excessive sweating at first and later, when the arterioles contract from endarteritis, the skin may be excessively dry.

Complete lesions of a peripheral nerve may be well illustrated by the results following section of the ulnar nerve at the elbow, where it is in an exposed position. There is lower motor neuron paralysis, that is, flaccid paralysis with rapid wasting, the electric reaction of degeneration, and microscopic degeneration of muscle fibers in the muscles supplied, namely the flexor carpi ulnaris, part of the flexor profundus, the two inner lumbricals, the short muscles of the little finger, all the interossei, and the adductors of the thumb. The hand rapidly becomes wasted, except for the muscles of the thumb, which are supplied by the median nerve (Fig. 86). The ring and little finger assume a characteristic position of extension of the first phalanx and flexion of the last two phalanges (see figure) owing to loss of the lumbricals. Blisters may form and the nails may fall off.

The sensory loss is most instructive. *Epicritic sensation* is lost for the anatomical distribution of the sensory branches of the ulnar nerve (see Fig. 86). Thus the whole area enclosed by the *broken* line in the diagram is insensitive to light touch, as tested after shaving the hairs by a delicate pencil of absorbent cotton, does not recognize two points of a compass applied simultaneously as two, and does not detect changes of temperature slightly above and below blood heat. Protopathic sense is lost over the area inside the dotted line. Here the pain produced by a pin prick is not

felt, nor excessive heat (skin pain), though the pressure may be recognized and deep pressure may produce pressure pain. Extreme degrees of heat and cold are not differentiated, but pressure with a pencil or pin head is felt readily. In the area between the broken and dotted lines the pain of a pin prick or very hot tube of water is more disagreeable than on normal skin, and a light scratch with the finger nail is felt to be decidedly disagreeable as soon as it crosses from healthy skin to the anæsthetic area. These peculiar sensations are felt because of the overlapping of nerves for protopathic sense from the median nerve.

Lastly, there is a narrow area along the medial border of the hand enclosed in the drawing within a continuous line where the skin is insensitive to all forms of stimuli, even to deep pressure with a pencil. The little finger too is probably insensitive to passive changes in position. But between the dotted and unbroken line deep pressure with a pencil is felt as pressure, and if heavy enough produces pressure pain; and the pressure of a heated test tube may be felt but not its hotness. This feeling of pressure over the area insensitive to protopathic sensory stimuli is owing to overlapping of deep nerves from the median.

If now the ulnar nerve be successfully sutured, in six or eight weeks protopathic sense will return over the whole anæsthetic area, but the sense of heat and cold and pain from pressure or pin prick will be of a diffuse character, and painful sensations will cause a more disagreeable feeling than in the healthy skin. In a year or more epicritic sense will return, and with its return protopathic stimuli will produce sensations which progressively approach the normal and localization will become more and more accurate. Vasomotor control returns with protopathic sense. Voluntary motion returns at about the same time as epicritic sense. If a considerable time elapses between the division and the repair of the nerve, the time before return of sensation and motion may be much prolonged and the chances of improving the condition by suture become less the longer the delay.

Effect of cord lesions involving sensory tracts. The peripheral sensory neurons which convey the *muscle-tendon-joint sense complex*, including the sense of weight and of passive position, and stereognostic sense, and also those which convey *bone sensibility* as tested by a tuning fork, and *compass sense* enter the posterior column (Fig. 69) and travel up in it on the same side till they reach the nucleus gracilis, if they come from the leg, or the nucleus cuneatus, if they come from the arm.

Posterior sclerosis. Occasionally in cases of profound anæmia one may get a sclerosis of the cord which is confined almost entirely to the

posterior columns, and especially to the long fibers of these columns. Figure 103 shows the position of the sclerosis in such a case recorded by Thompson, *Brain*, Vol. XXXIV, page 511. This case will be discussed fully later, but for the present it suffices to say that the patient had no loss of motor power, but his hands were awkward. He could not touch his nose when his eyes were closed, and had difficulty in walking in the dark, that is, he was ataxic owing to his loss of muscle sense. He had no loss of the senses of heat, cold, pain, light touch or pressure, but he had a profound loss of the sense of passive position, especially of the hand and fingers; if when his eyes were shut the physician moved his fingers, forearms, or legs, he could not tell where they had been placed. He could not tell, unless he could see the object, that a pound was heavier than half a pound. He could not tell if he were touched by one point of a compass or by two points simultaneously. Tuning-fork sense was not tested, but it was probably lost. This and allied cases prove that the long fibers of the posterior columns convey conscious sense of passive position, sense of weight, and tactile discrimination (compass sense), and the groups of muscle-tendon-joint sense impressions that underlie the power of maintaining static equilibrium; also they convey the sense of texture and bone sensibility as tested by a tuning fork placed against a subcutaneous bony prominence. They do not convey pain, heat and cold. In most cases they convey light touch and pressure, forming one of the two routes by which these sensations usually travel, though in the special case quoted these sensations were probably entirely relayed, crossed in the commissure of the cord and traveled upward by the ventral spinothalamic tract (Fig. 69).

Second sensory neuron for muscle-tendon-joint sense, etc. The second sensory neuron for this particular group of sensory impulses is supplied by the opposite medial lemniscus (Figs. F to K). The crossing takes place in the medulla oblongata (Fig. F).

Figure 174 shows a lesion of the left medial lemniscus which caused loss of the sense of passive position and loss of tuning-fork sense over the right side of the body with ataxia of the right arm and leg. It will be seen that this patient also had a lower motor neuron paralysis of his left twelfth nerve, which was involved in the lesion, and a spastic (upper motor neuron) paralysis of the right side of his body from involvement of the pyramidal tract. Figure 178 illustrates an analogous case with a lesion in the pons.

The lowest sensory neurons for pain, heat and cold, as has been seen, are probably unmyelinated and enter the posterolateral column of the cord in which they bifurcate and run up and down for a short distance, sending collaterals into the gelatinous substance. Hence in localized lesions of the cord involving the posterolateral tract we may have loss of pain, heat and cold with a homolateral segmental distribution to the extent of the lesion. These symptoms are mixed with others of a more remote character. This variety of lowest sensory neuron anæsthesia is comparatively rare in lesions of the cord, but is peculiarly frequent in the case of disease involving the spinal tract of the trigeminal nerve. (See Fig. 179.)

Second sensory neuron for pain, heat and cold. The lowest sensory neurons for pain, heat, and cold are entirely relayed within 2 to 6 cm. above their level of entry by secondary neurons whose cell bodies are in the gelatinous substance or in the gray matter in its immediately neighborhood. These neurons cross in the white or more probably in the gray commissure (decussation) of the cord and ascend in the posterior spinothalamic tract. Because of its location the course of this neuron in the anterior commissure and in the posterior gray column, where its cells are placed, is peculiarly liable to interruption in *syringomyelia,* and therefore one of the earliest symptoms of syringomyelia is a segmental loss of pain, heat and cold without loss of other forms of sensation (Figs. 111, 224-b, 9,9′, 112, 187, 215 and 216).

The position of the posterior spinothalamic tract in man is demonstrated by such lesions as the one described in the University of Pennsylvania Medical Bulletin, July and August, 1905, by Spiller. In this case (Fig. 104, a and b) the posterior spinothalamic tract on each side of the cord was cut across by two separate small tuberculous foci in the thoracic portion of the cord almost as accurately as by a physiological experiment. All other tracts escaped. The effect was that the patient had lost all sense of pain and temperature in his legs without any other motor or sensory loss whatever.

Figures 176 and 179 illustrate destructive lesions of the posterior spinothalamic tract in the medulla oblongata and in the pons; the medial lemniscus has escaped. In both cases there is loss of pain, heat and cold for the opposite side of the body without loss of the other forms of sensation. In Figure 176 there is illustrated an interesting form of crossed anæsthesia, due to a lesion involving the spinal tract of the fifth nerve

and the neighboring spinothalamic tract. There results loss of pain, heat and cold for the face on the same side as the lesion, but for the body the loss is on the side opposite to the lesion.

Figure 85 illustrates a case in which a knowledge of the position and function of this tract led to a successful operation for the relief of pain produced by inoperable pelvic cancer. The patient had cancerous glands involving the sacral plexus on the right side and causing great pain referred to the right leg. Section of the posterior spinothalamic tract on the left side of the spinal cord in the region of the lumbar enlargement gave complete relief from pain without interference with the functions of the leg.

As the second sensory neurons for pain, heat, and cold cross in the cord, while those for muscle-tendon-joint sense cross in the medulla oblongata, a half-section of the cord (Figs. 109 and 110) results in loss of pain, heat and cold for the opposite side of the body below the lesion, with loss of muscle-tendon-joint sense, tuning-fork and compass sense on the same side as the lesion and below it. Hemi-section of the cord also produces upper motor neuron paralysis on the same side as the lesion. This combination of symptoms is known as Brown-Sequard paralysis.

Touch and pressure. As simple touch tested by a light fluff of absorbent cotton and also pressure tested by a pencil point or the head of a pin both take an inconstant course through the cord, these forms of sensation are of little value in testing cord lesions, though they are of great value in the diagnosis of diseases of the peripheral nerves. Clinically it would appear that in some cases both these forms of sensation are quickly relayed by secondary neurons whose cells are in the posterior gray columns and whose axons cross in the white or gray commissure of the cord and ascend in the ventral spinothalamic tract. In other cases the path seems to lie in the posterior column as far as the medulla oblongata, where it is relayed into the opposite medial lemniscus (Fig. 69). In most cases they appear to travel by both routes. By whatever route these forms of sensation ascend in the cord, they reach consciousness in the medial nucleus of the thalamus of the opposite side from that of their peripheral origin.

THE THIRD SENSORY NEURON

We now have to deal with the function of the thalamus, the essential thalamic organ, and the thalamocortical neuron.

Excluding the motor area, the visual area in the occipital lobe, the auditory area in the superior temporal convolution, and the olfactory area in the uncinate extremity of the gyrus hippocampi, all of which give defi-

nite reactions to experimental methods in monkeys and in lower animals, extensive experimental lesions of the other parts of the cerebral cortex have failed to cause any appreciable motor or sensory loss. Sensory loss has occurred in lesions of the gyrus cinguli (Ferrier), but this has been ascribed by Schaefer to possible damage to lower cerebral structures. Clinical cases, however, carefully examined by neurologists, seem to furnish the interpretation of this absence of sensory loss in cortical lesions. Tactile sense, pain thermic sense, the sense of texture, and tuning-fork sense all seem to reach consciousness in the thalamus. I judge this because stationary cortical lesions in man which cause sensory disturbance do not give rise to complete loss of these sensations but to errors in judgment regarding their intensity, nature, and localization.

Most lesions of the thalamus are vascular, either hæmorrhagic or ischæmic; the latter are due to thrombosis or embolism of the basilar arteries, causing softening. Gross lesions, if they are not fatal, cause profound loss of all forms of ordinary sensation and may be indistinguishable from lesions in the subthalamic region involving the medial lemniscus. If the internal capsule be also involved, there will be concurrent hemiplegia; both the hemianæsthesia and hemiplegia affect the opposite side of the body and include the face. If the optic radiations or pulvinar(?) be included there will be hemianopsia.

Limited lesions of the thalamus may cause loss of one or more forms of sensation, other forms escaping (Head and Holmes, *Lancet*, 1912). Sense of posture suffers most; tactile sense is frequently diminished, but seldom abolished; compass sense is dulled, in order to be recognized as two the points must be further apart than on the normal side; thermic sense is lost or much altered; vibratory sense is seldom lost; and the sense of roughness is not much affected. There are some cases of limited thalamic lesions characterized by considerable sensory loss which is associated with acute pains, persistent, paroxysmal, often intolerable, yielding to no analgesic. There are frequently choreic and athetoid movements. There is also frequently a tendency to react excessively to pleasant or to unpleasant stimuli. Head and Holmes believe this last symptom is due to interruption of the thalamocortical fibers providing for cortical control of thalamic reactions (Fig. 220).

The fourth or thalamocortical sensory neuron. The thalamocortical sensory neuron leading to the parietal lobe provides for cortical motor responses, for the registration of sensory impressions in the form of memories, for sensory judgments, and for the whole group of cortical reactions

to peripheral sensory stimuli that take so large a part in cerebral activities. Errors in judgment as to the localization of stimuli or as to differences in the weight of objects, mistakes in differentiating degrees of roughness and smoothness or of heat and cold, these symptoms accompanied in many cases by ataxia of the opposite limbs and by tactile agnosia (astereognosis) are the chief characteristics of subcortical and cortical lesions in the parietal lobe, especially the posterior central convolution and neighborhood of the meeting of the superior post-central and intraparietal sulci (Fig. 142). As the extremities of the limbs are most highly educated, sensory loss due to a cortical lesion is always most marked in the forearm and hand, lower leg and foot (Fig. 221). No stationary cortical lesion, experimental or clinical, causes complete loss of the primary forms of sensation except during the period of shock immediately following the injury.

Visceral sense for spinal nerves. While it is known that visceral sense travels by spinal (or cranial) sensory nerves which pass uninterrupted through the sympathetic ganglia and enter the spinal cord by way of the posterior nerve roots, nothing is known of the tracts in the cord for visceral sensation. It is certain that visceral pain is conveyed to the thalamus by way of the posterior spinothalamic tract.* Undoubtedly visceral reflexes are provided for in the spinal cord and visceral sense reaches consciousness in the essential thalamic organ, whether it be a sense of well-being or discomfort. Visceral sensory judgments are probably dependent on a relay from the thalamus to the parietal cortex, and perhaps thence to the frontal lobe.

Nerves of Special Sense. So far we have considered all those sensory nerves and their central tracts which convey the various forms of ordinary bodily sense. It is necessary now to deal with the nerves of special sense. These are:

1. The sensory portion of the *vagus* (tenth nerve). With the exception of its auricular branch, and those sensory branches which supply the pharynx and larynx, the other sensory branches of the vagus (œsophageal and gastric, tracheal, pulmonary and cardiac) convey visceral sense.

2. The *glossopharyngeal* contains fibers of ordinary sense for the back of the tongue, middle ear, soft palate and upper part of the pharynx. In addition it is the special nerve of taste for the posterior third of the tongue.

* This has lately been proved by section of both posterior spinothalamic tracts in the upper thoracic region for the relief of the intolerable pain of gastric crises in tabes dorsalis. The operation has proved successful in many cases.

3. With the gustatory portion of the glossopharyngeal should be described the gustatory portion of the chorda tympani, which is the nerve of taste for the anterior two-thirds of the tongue.

4. The vestibular portion of the eighth nerve is concerned with the special sense of equilibrium. It is so closely related to the cerebellum that it will be described with the cerebellar connections.

5. The cochlear portion of the eighth nerve is the nerve of hearing.

6. The second or optic nerve is the nerve of sight.

7. The first or olfactory nerve is the nerve for smell.

These will now be considered in the order in which they have been enumerated.

The afferent portions of the tenth nerve (vagus or pneumogastric). All the afferent neurons of the vagus have their cell bodies in the jugular and nodose ganglia. They are divisible into somatic and visceral afferent. The afferent fibers for the skin of the auricle and external auditory meatus, the middle ear(?), the pharynx, and larynx may be classed as somatic afferent; those for the trachea, lungs, œsophagus, stomach, and heart as visceral afferent.

The vagus possesses *one cutaneous branch called the auricular branch.* It is distributed to the skin of the external acoustic meatus and back of the auricle. Centrally it probably joins the spinal tract of the fifth nerve. It appears to furnish the anatomical basis for the hacking, dry cough which is occasionally caused by irritation of the external auditory meatus as by a mass of inspissated wax.

The central axons of the branches of the vagus which convey the sense of *touch, pain, heat and cold from the pharynx and larynx* probably also joint the spinal tract of the fifth nerve as the entering vagal roots pass through this (Figs. 59, F, 14 and 15). These will be relayed by the opposite trigeminothalamic tract.

The rest of the afferent fibers of the vagus are *visceral afferent* from the trachea and lungs, œsophagus and stomach, and heart. Their central axons end in the dorsal nucleus (Figs. 59, F, 14 and 15). Nothing is definitely known of the relays for visceral sensation or its central representation for the visceral afferent nerves in either the cord or the brain stem, and the subject is too complex for treatment here.

The afferent fibers of the ninth or glossopharyngeal nerve are of two classes, those which convey ordinary sensation and those which convey the sense of taste. The parent cells of the fibers which convey ordinary sensation are in the superior and the petrous glossopharyngeal ganglia.

The somatic afferent branches convey sense of touch, pain, heat, and cold from the pillars of the pharynx, front of the epiglottis, posterior part of the tongue, and glossoepiglottic folds. They probably join the spinal tract of the fifth nerve. Visceral afferents with cells in the same ganglia come from the middle ear. The classification of these is doubtful.

The peripheral endings of the *gustatory fibers* of the glossopharyngeal nerve are in special taste organs situated in the posterior third of the tongue and in the fauces, tonsils, back of the pharynx and lingual surface of the epiglottis. The central axons enter the fasciculus solitarius, ending in its nucleus. On physiological evidence this nucleus, which is now regarded as the gustatory nucleus, must be abundantly connected by association neurons with a wide range of efferent neurons. Witness the great range of taste reflexes varying from simple alterations in salivary secretion to reactions involving the whole mechanism of vomiting. A lesion of the *fasciculus solitarius* may cause complete loss of taste for the corresponding half of the tongue. The paths by which the sensory neurons for taste are relayed centralward are altogether unknown. The cortical center is probably in the uncus of the gyrus hippocampi or gyrus cinguli.

The gustatory fibers of the chorda tympani nerve end peripherally in the anterior two-thirds of the tongue; their parent cells are in the geniculate ganglion of the facial nerve. Their central axons join the fasciculus solitarius.

The balance of evidence at present seems to indicate that loss of taste after destruction of the trigeminal nerve by operation or disease is due to consequent trophic changes in the tongue interfering with the taste reactions of the gustatory end organs, and is not due to any taste fibers contained in the trigeminal sensory root. On this subject clinical observations after excision of the semilunar ganglion (on the sensory root of the fifth nerve) are very conflicting (see *Brain,* Vol. XXX, p. 219).

The cochlear division of the eighth nerve (Figs. 102 and 61) is the nerve of hearing. The ganglionic cells of the cochlear nerve form the spiral ganglion which occupies the canalis spiralis cochleæ. Its peripheral processes are distributed to the hair cells of the spiral organ of the cochlea. The central axons of these cells form the cochlear nerve, which enters the brain stem lateral and slightly caudal to the vestibular nerve, in the floor of the lateral aperture of the fourth ventricle (Figs. 10, 11, 12, 13, 102 and 61). The axons end in the ventral and dorsal cochlear nuclei (Figs. 102, 61), where the first relay takes place.

Most of the axons of the ventral nucleus (Fig. 102) pass transversely

through the pons between the basilar and tegmental portions, forming the corpus trapezoideum (Figs. H, I, 63 and 102). Many of these are interrupted by cells in the superior olive or by scattered cells of the nucleus of the corpus trapezoideum of the same and the opposite sides. After this second relay the corpus trapezoideum takes an upward (cephalad) direction in the immediate neighborhood of the superior olive of the side opposite to that of the nerve entry, and a little higher in the pons it becomes the lateral lemniscus (Fig. J). The fibers which are interrupted in the dorsal nucleus (Fig. 102) are relayed by the cells of this nucleus, and the axons of the dorsal nucleus, together with some axons from the lateral part of the ventral nucleus, form the striæ medullares (Figs. 11, 61 and 102). These cross medialward, some pass to the superior olive of the same side; others cross the floor of the ventricle, plunge into the medial sulcus and join the opposite superior olive, or become directly longitudinal in its neighborhood, and join the lateral lemniscus without interruption. The lateral lemniscus at first lies lateral to the medial lemniscus in the upper part of the pons; higher up it shifts to the lateral side of the brachium conjunctivum (Fig. J). Here another nucleus is found called the nucleus of the lateral lemniscus. In man and orangs this has been shown to be continuous with the superior olive. The lateral lemniscus sends terminals or collaterals to the inferior colliculus (Fig. K) and ends in the medial geniculate body (Fig. L). From the medial geniculate body the auditory path is relayed to the auditory center in the posterior third of the superior temporal convolution and the anterior transverse temporal gyrus (Figs. 90 and 92). This relay forms the auditory radiation (Fig. M). In the immediate neighborhood of this primary acoustic area is the secondary acoustic area (Figs. 90 and 92) where word memories are stored; the secondary acoustic area is on the left side in right-handed people.

Short reflex path from auditory nuclei to eye-turning muscles. It has been seen that certain of the impulses received by the cochlear nerve pass by means of a relay to the superior olive of the same side. The superior olive (Figs. H, 63 and 102) is united to the nucleus of the sixth nerve by many prominent association neurons which together form the peduncle of the superior olive. The sixth nucleus supplies the external rectus of the eyeball, and controls the opposite internal rectus through association neurons which pass from the sixth to the third nucleus of the same or of the opposite side (see third nerve) by way of the medial longitudinal fasciculus. There is thus provided a short reflex mechanism

by which the eyes can be directed toward the side from which a sound proceeds. There are probably also association fibers descending by the medial longitudinal bundle to the nuclei of the accessory and first cervical nerves and forming a reflex arc for turning the head toward the direction from which a sound is heard.

Guddens' Commissure. In the posterior part of the optic chiasm and in the optic tracts there are commissural fibers which unite the medial geniculate body of one side to the opposite inferior colliculus. The connections of this commissure establish it as a part of the auditory mechanism, but its physiological significance is unknown.

Physiology. An incomplete lesion of one cochlear nerve causes defective hearing in the involved ear with tinnitus. Complete destruction of the nerve causes total unilateral deafness. There is usually simultaneous involvement of the vestibular nerve and often also of the facial nerve. Disease of the nerve fila in the brain stem would be likely to include the restiform body with consequent cerebellar symptoms. Interruption of the lateral lemniscus or of the auditory tracts or centers above this does not cause absolute deafness of the opposite ear because some of the fibers of the lateral lemniscus are uncrossed. This is the general opinion, though Dejerine states that all the fibers are crossed and that, therefore, total deafness of the opposite ear results from a complete lesion of the lateral lemniscus. The branchium conjunctivum is likely to be involved in affections of the lateral lemniscus with resulting cerebellar symptoms (Fig. J).

Involvement of the auditory radiation (Fig. M) causes partial deafness of the opposite ear (complete if the lesion be complete according to Dejerine). The motor tract in the basis pedunculi (Fig. M) is likely to be involved with hemiplegia of the opposite side. Cortical deafness due to lesion of the superior temporal convolution of one side is never complete owing to bilateral representation of each ear. (It may be complete according to Dejerine.) The opposite ear is chiefly represented in the superior temporal convolution. An affection of the secondary auditory area (Figs. 92 and 142) which lies on the left side in right-handed people may cause word deafness (sensory aphasia). If extensive, this may be accompanied by other forms of aphasia and by intellectual deterioration. (See aphasia.)

The visual paths (Fig. 105). In the retina the rods and cones are the sensory end organs. The bipolar cells which connect with these collectively form the true morphological optic nerve; the cells of the gangli-

onic layer furnish the cell bodies of the neurons of the first relay of the visual paths. Thus the axons of the ganglionic cells of the retina form the optic nerve of anatomical nomenclature. At the optic chiasm a decussation takes place. In man and in those mammals that have binocular vision the decussation is partial; the fibers from the nasal half of each retina intercross, while those from the temporal half of each retina join the optic tract of the same side (Fig. 105). Thus each optic tract carries fibers from its own half of both retinæ (see Fig. 105), and as the light rays cross in the lens each optic tract bears light impressions from the opposite half of the field of vision. The fibers from the center of vision (macula lutea) of both eyes are represented in both optic tracts. In the posterior part of the optic chiasm and in each optic tract there are fibers which do not belong to the eyes at all, but which unite the corpora geniculata interna with the opposite posterior colliculi. From their relations these must be auditory in function; they have been described with the connections of the cochlear nerve. Their use is unknown.

The visual part of the optic tract as it is traced backward is found to end in the lateral geniculate body (Figs. 105, L and 68); some fibers pass by the lateral side of the lateral geniculate body to go to the pulvinar of the thalamus, and others go to the superior colliculi, mainly that of the opposite side. These fibers form a distinct layer, the stratum opticum, on the surface of the superior colliculus.

From the cells of the pulvinar and lateral geniculate body spring the fibers of the last relay. Together these fibers form the optic radiation (Figs. 87, b and 105) which passes to the cortex of the calcarine fissure and the neighboring area of the cuneate and lingual gyri (Figs. 90 and 93). This is the primary cortical center for vision. It will be apparent that it receives macular and homonymous fibers from both retinæ, that is, it receives fibers from both maculæ luteæ and fibers from the half of each retina which corresponds to its own side. This area of the cortex, owing to its peculiar naked-eye characters, is called the area striata. It is frequently also called the calcarine area. The calcarine area receives no fibers from the superior colliculus, but sends fibers to it. The cortex of most of the occipital lobe has a secondary visual function, that of storing visual memories. Especially is this true of the left occipital lobe in right-handed people.

Physiology. The *superior colliculi* have no direct visual function. They appear to coördinate the movements of the eye muscles and probably of the whole body musculature with visual impressions, though in higher

mammals they may be subjected to extensive experimental injury with very little result, and that little extremely temporary. Though of vital importance in fishes and birds, in mammalia and especially in the higher mammals they appear to be largely subordinated to higher centers. A pure lesion of the *superior colliculi* in man has as its distinctive symptom paresis of the upward and downward movements of the eyes. Lateral movements are so much dominated by the cortex cerebri and Deiters' nucleus that they are unaffected. It is probable that a limited lesion of the *pulvinar* gives rise to no symptoms if the lateral geniculate body and optic radiation escape.

A complete lesion of the *lateral geniculate body* causes homonymous hemianopsia on the side opposite the lesion. This is because all visual fibers of the optic tract are relayed here on their way to the cortex.

Figure 106 shows what is meant by the visual fields. Within the continuous line is the whole field for white light for each eye. As the nose interferes with light rays passing to the temporal or outer half of each retina, it will be noted that the nasal or inner half of each *visual field* is the smaller. Owing to the crossing of rays of light in the lens, the inner or nasal half of the field of vision is that which corresponds to the outer or temporal half of each retina. Thus while only half the fibers of the optic tract are crossed, yet each tract represents a complete crossing of one-half of the visual field. A lesion of the left optic tract, therefore, causes blindness of the right half of the field of vision for both eyes, or right homonymous hemianopsia. This is rendered clear by Figure 105.

To comprehend the following description one must remember that the blindness refers to the field of vision and is always referred to the side opposite to the lesion. Reference to Figure 105 will make the description intelligible.

1. Complete destruction of one optic nerve (for the sake of clearness let us say the left), as at *1*, Figure 105, will cause blindness of the same (left) eye. A partial lesion of one optic nerve in front of the chiasm gives rise to a central scotoma (blind spot).

2. A lesion at *2* destroying the fibers lateral to the chiasm on the left side will cause blindness of the temporal half of the left retina, or nasal hemianopsia of the left eye; the center of the visual field will escape.

3. A lesion limited to the decussating fibers of the chiasm (Fig. 105, 3) is rare, but may occur. It causes bi-temporal hemianopsia, due to blindness of the nasal half of each retina. Again the macula escapes. It is usually associated with infundibular or pituitary symptoms.

4. A complete lesion of the left optic tract causes right homonymous hemianopsia (Fig. 107) due to blindness of the left half of each retina; central vision escapes.

5-5′. A lesion of the left lateral geniculate body or pulvinar has the same effect as *4*, except that both lesions may be accompanied by other thalamic or capsular symptoms (hemianæsthesia and perhaps hemiplegia), depending upon the extent and distribution of the lesion.

6. A limited lesion of the left optic radiation causes the same symptom as *4*. Cases *4*, *5*, and *6* can only be distinguished from each other clinically by accompanying symptoms due to lesions of neighboring structures. Wernicke's pupillary reaction, described below, is not reliable.

Wernicke's hemiopic pupillary reaction (Fig. 105) is absence of pupillary contraction when a ray of light is thrown on the blind side of the retina. The contraction of the pupil to light depends on afferent impulses reaching the superior colliculus by way of the optic tract; the efferent path is by way of the third nerve and ciliary ganglion. If the lesion causing hemianopsia be above the optic tract (*i.e.*, in the optic radiation or calcarine area), the afferent path will escape and a ray of light thrown on the blind side of the retina will cause contraction of the pupil; but a lesion in the optic tract will interrupt the afferent portion of the reflex arc, and the pupil will then *fail to contract* when a ray of light is thrown on the blind side of the retina.

7. A left cortical lesion of the calcarine area causes blindness of the left half of each retina, right homonymous hemianopsia; however, this is seldom as complete as it is in any of the previous lesions. If the lesion be limited to the area above the left calcarine fissure the blindness will be limited to the left upper quadrant of each retina. There will result right homonymous quadrantal blindness of the right *lower* quadrant of each field of vision. Lesions below the calcarine fissure cause upper quadrantal blindness of both visual fields (Fig. 108). A bilateral lesion of the calcarine area causes cortical blindness in which the whole visual field for both eyes is obscured or blind, according to the degree of the cortical destruction; but even in bilateral cortical blindness central vision is seldom completely lost.

8. A lesion at *8* in the left hemisphere in a right-handed patient includes:

(*a*) The fibers of the left optic radiation.

(*b*) Fibers from the area striata to the angular convolution where visual word memories are stored.

(*c*) Fibers from the opposite area striata by way of the splenium of the corpus callosum.

The result of such a lesion is right homonymous hemianopsia due to *8a;* complete loss of visual word memories due to *8b* and *8c*. The consequence is that though he is able to see written or printed characters, the patient has lost all power of interpreting them. He has lost his memories of printed and written words. He may hear, speak, and do copy-writing, but he cannot read even what he has copied.

In hemianopsia, patients having central vision unaffected are not infrequently unconscious of their defect of vision which has to be carefully searched for by plotting the visual fields; so the patient with the lesion at *8* might only complain that he had lost the power of reading. This is known as pure word-blindness (alexia).

9. A cortical lesion of the angular convolution on the left side in a right-handed man may cause alexia (word-blindness) without other visual defect. It is usually accompanied by other speech defects.

For further discussion of this see aphasia.

The olfactory nerve. Though of the utmost interest phylogenetically, the olfactory mechanism has a very limited clinical importance, as man is very little guided by smell in his daily life.

The olfactory area of the nose is limited to a small portion of the upper part of the septum and upper concha. Here the olfactory epithelial cells are imbedded in the mucosa. These cells are at once the end organs for smell and the cell bodies of the nerves; in this respect the olfactory nerve approaches more closely than any other nerve in vertebrates the primitive type of peripheral sensory neuron. The central processes of the olfactory cells penetrate the cribriform plate of the ethmoid and enter the olfactory bulb where the first relay takes place. The further connections are exceedingly complex, and will not be detailed. All that need be noted here is that the ultimate termination is in the pyriform area of the hooked extremity of the hippocampal gyrus (Fig. 93). The hippocampus, fornix, corpora mammillaria, mammillothalamic bundle, the anterior tubercle of the thalamus, part of the anterior commissure, the nucleus habenulæ, the tænia thalami, all belong to the olfactory nervous mechanism, but they acquire significance only when examined in the light of comparative anatomy.

Clinically, olfactory nervous connections are not crossed. A lesion of the olfactory bulb or tract involves loss of the sense of smell of the corresponding side of the nose. Subjective sensations arising spontane-

ously of an odor, usually offensive, may point to a tumor in the neighborhood of the piriform area.

THE VESTIBULAR APPARATUS AND THE CEREBELLUM, THEIR CONNECTIONS AND FUNCTIONS

For the complete connections of the cerebellum, see Figure 121.

The most primitive cerebellum as seen in amphibia, reptilia, and some bony fishes is little more than the head ganglion of the vestibular nerve, and even in its highest development the cerebellum retains its intimate relations to this nerve, though its muscular and ocular connections in mammalia outweigh its vestibular relations. Unlike the rest of the central nervous system, there is no intimate commissural connection between the parts of the cerebellum; the vermis and hemispheres are each independent.

Vermis. With the exception of the flocculus, which is rudimentary in man, the vermis is phylogenetically the oldest part of the brain. It consists of a cortex, central nuclei, the nuclei tecti and nuclei globosi (Fig. 60) and afferent and efferent pathways. The nucleus emboliformis and nucleus dentatus are more intimately connected with the cerebellar hemisphere. The chief afferent nerve is the vestibular nerve and the semicircular canals may be regarded as especially the peripheral organ of the vermis. But afferent paths from all the trunk muscles also reach the vermis.

The vestibular nerve (Figs. G, 60, 61, 121, and 128). The ganglionic cells are bipolar and lie in the trunk of the vestibular nerve in the internal acoustic meatus. The peripheral processes end in connection with the hair cells of the maculæ in the utricle and saccule and in the ampullæ of the semicircular canals. The axons form the central portion of the vestibular nerve which enters the upper part of the medulla oblongata at its junction with the pons (Fig. G). The fibers pass dorsally between the spinal tract of the fifth nerve and the restiform body (Figs. G, 60 and 61) and end in three nuclei which lie in the immediate neighborhood of the acoustic tubercle (Fig. 11). A large descending tract, the spinal tract of the vestibular nerve is formed of many bundles of fine fibers which are readily distinguished in the upper part of the medulla oblongata (Figs. G, 60 and 61). The spinal tract is surrounded by gray matter containing small nerve cells. Of the three nuclei the dorsal nucleus is triangular in section, and lies in the floor of the ventricle (Figs. 60 and G). Its cells are small. The nucleus of Bechterew is rather higher and more

laterally placed and is medial to the brachium conjunctivum (Fig. H). Its cells are large. The nucleus of Deiters (Fig. G) consists of many large multipolar cells scattered among the terminal fibers of the vestibular nerve ventrolateral to the dorsal nucleus. It is said that some of the fibers of the vestibular nerve pass to the nucleus fastigii (see Fig. 128). Deiters' nucleus is in afferent relation with the somatic muscles by collaterals from the spinocerebellar tracts. For the conscious knowledge of the position of the head there must be a crossed tract to the thalamus(?) and cortex, perhaps the temporal lobe (middle and inferior temporal gyri—Mills).

The vestibular efferent pathway though reinforced from the vermis and roof nuclei of the cerebellum is so largely independent that it may be better to describe it here. The *cells of Deiters'* nucleus form three distinct efferent pathways to the lower motor neurons (Figs. G and 128).

1. Fibers pass directly to the nucleus of the sixth nerve on the same side, and by way of the medial longitudinal bundles to the third and fourth nuclei, mainly to those of the opposite side (Figs. G and 128). Thus connections are established with the eye muscles.

2. Other efferent fibers pass to the lower motor neurons of spinal nerves, traveling by way of the medial longitudinal bundle, mainly that of the same side. Below the oblongata these fibers are found in the anterior white column of the cord.

3. Fibers also pass to the lower motor neurons of spinal nerves by the homolateral vestibulospinal tract (Figs. 121 and 128). This tract lies medial to the spinal tract of the fifth nerve in the medulla oblongata, and along the anterior border of the cord lower down (Figs. 69, G to A). The vestibulospinal tract is joined in the neighborhood of Deiters' nucleus by fibers from the nuclei tecti (chiefly of the opposite side), the nucleus globosus and the nucleus emboliformis(?). (Figs. 128 and 121.) (Thomas.)

Physiology. The saccule, utricle and semicircular canals form the special organ for orientation in space. That the whole body may be kept in equilibrium this vestibular apparatus must be able to control the movements of the eyes so that they change their position with every alteration in the position of the head. The mechanism for equilibration must also be able to maintain a *tonic* and *coördinating action* over all the body muscles. This it does through the efferent tracts described.

In sharks, section of the vestibular nerves causes great depression of muscle tone. "At Naples Ewald saw some sharks which required four vigorous arms to hold them, and were held by a single hand after section of the auditory (vestibular) nerves; nevertheless after the removal of

large portions of the cerebellum, which constituted a much graver operation, this muscular enfeeblement did not take place." In mammals the loss of tone after bilateral section of the vestibular nerves is very transient, but section of the vestibulospinal tracts in monkeys causes more marked paralysis than section of the pyramidal tracts. This is probably due to the fact that in mammals the vestibulospinal tract represents both a vestibular and a cerebellar pathway. The vestibular apparatus is *an organ for equilibration and for the maintenance of muscle tone.*

The paths from peripheral muscles to the vermis. The vermis is in touch through afferent pathways with every striped muscle in the body.

1. *The dorsal spinocerebellar tract* (Figs. B to G; see also 121). Nerves carrying muscle sense very soon after entering the lateral side of the posterior column send terminals or collaterals to the nucleus dorsalis (Figs. B and 69). This nucleus extends from the eighth cervical to the second lumbar segment of the cord. It probably receives peripheral afferents from the upper thigh and trunk muscles only. The axons of the large cells of the nucleus dorsalis pass lateralward in the cord to form the dorsal spinocerebellar tract (Figs. 69 and B). In the medulla oblongata this tract shifts backward and forms the central portion of the restiform body (Fig. G). It contributes collaterals to the formatio reticularis and perhaps to the nucleus lateralis of the medulla oblongata, to Deiters' nucleus, and to the corpus dentatum, and it ends in the cortex of the vermis after its fibers have largely crossed the middle line.

2. *The ventral spinocerebellar tract* (Figs. 69 and B) myelinates later than the dorsal, and commences lower down in the cord (Figs. 50, 51, 52 and 54). Afferent nerves from leg muscles after entering the posterior column arborize round cells somewhere in the posterior gray column (Fig. 69). The axons of these cells form the ventral spinocerebellar tract. (Gowers' column of old terminology includes the ventral spinocerebellar tract, the dorsal spinothalamic tract, and spino-olivary and spinotectal endogenous neurons.) The ventral spinocerebellar tract may be partly homolateral, partly crossed. It appears to receive accessions from all the thoracic segments, and also, according to McNally and Horsley, from the cervical region. In the oblongata it contributes collaterals to Deiters' nucleus, to the cerebellar hemispheres, and to the corpus dentatum; and it appears on the surface of the brachium conjunctivum in the isthmus (Fig. I). Curving round the brachium conjunctivum it passes to the vermis by way of the superior medullary velum, sending collaterals to the corpora quadrigemina through the frenulum veli.

Cerebellar fibers from the arm muscles may reach the vermis through accessions to the spinocerebellar tracts in the cervical cord (McNally and Horsley, *Brain,* Vol. XXXII), or through the accessory cuneate nucleus (André Thomas and others) by fibers from this nucleus to the restiform body. The lateral sensory nucleus of the fifth nerve is the probable source of cerebellar fibers from most of the cranial nerves, except the second and eighth. The fibers join the restiform body, or according to some neurologists, may pass directly from the fifth nerve by the medial side of the brachium conjunctivum to the vermis. I doubt very much whether any fibers pass directly to the cerebellum from any sensory nerve without a relay in the cord or brain stem. The *optic nerve* may be connected with the vermis by fibers descending from a relay in the superior colliculi through the brachium conjunctivum to the corpus dentatum or nucleus fastigii or both. The cochlear nerve may have similar connections.

The cerebellar hemispheres appear first in the mammalia and develop in direct ratio with the increasing importance of the cerebral cortex, reaching their greatest size and complexity in man. With these, the inferior olives also increase in size. Each cerebral hemisphere bears a crossed relationship by cortico-ponto-cerebellar neurons to the opposite hemisphere of the cerebellum and the inferior olive has a somewhat similar relation. When for any reason one cerebral hemisphere fails to develop or undergoes marked gross degeneration, a similar defect in development or atrophic change occurs in the inferior olive of the same side and the cerebellar hemisphere of the opposite side, along with the corresponding brachium pontis and peripheral olivary fibers of the restiform body (Fig. 84). The vermis on the other hand has no direct relationship with any part of the telencephalon, unless perhaps the occipital lobes and rhinencephalon, the cerebral cortical centers for smell and vision.

Corticocerebellar fibers. The frontal lobe sends efferent fibers through the anterior limb of the internal capsule (Fig. 87, e) and medial fifth of the basis mesencephali (Fig. L) to the basilar portion of the pons. Here a relay takes place through the cells of the nuclei pontis by fibers which join with others to be described to form the opposite brachium pontis (Figs. I and 121). The temporal lobe (perhaps also the parietal and occipital lobes) sends fibers by the posterior part of the internal capsule (Fig. 87, e) and lateral fifth of the basis mesencephali (Fig. L) to the basilar portion of the pons, where they are relayed in the same way as the fronto-pontine fibers just described.

Finally the pyramidal tracts as they pass through the pons (Fig. I)

contribute collaterals to the nuclei pontis, and thus bring the anterior central convolution into relation with the opposite cerebellar hemisphere. The brachium pontis therefore consists of axons of the cells in the opposite basilar portion of the pons relaying most, perhaps all, parts of the opposite cerebral hemisphere. Its fibers radiate freely to all parts of the cerebellar hemisphere. The anterior external arcuate fibers and their nucleus belong to the cerebro-ponto-cerebellar system.

The inferior olive increases in size, fails to develop, or atrophies in direct ratio with the cerebral cortex of the same side and the cerebellar hemisphere of the opposite side (Fig. 84). It is a relay station to the opposite cerebellar hemisphere.

AFFERENT TRACTS TO THE INFERIOR OLIVE

Spino-olivary fasciculus. Spinal endogenous neurons with cell bodies in the anterior gray columns and axons ascending in the anterolateral white columns (see Fig. 78) end in relation with the dorsal surface of the inferior olive and the dorsal accessory olive.

The central tegmental tract is believed to end in the inferior olive. It is uncertain whether it comes from the thalamus or the corpora quadrigemina; perhaps it comes from both and thus forms an afferent tract from the great sensory nucleus, and from the coördinating centers for visual and auditory tracts to the inferior olive and so to the opposite cerebellar hemisphere.

The olivocerebellar bundle leaves the hilum of one olive, crosses the median line (Figs. 121, G, and 60), streams through or around the opposite olive without interruption, passes through or ventral to the spinal tract of the fifth nerve, and joins the restiform body of which it forms the peripheral fibers. The fibers end in all parts of the cortex of the cerebellar hemisphere of the side opposite to that from which they arise. Perhaps a few fibers are contributed to the vermis and nucleus dentatus.

Cerebellar efferent tracts. The axons of the Purkinje cells in the cerebellar cortex (Figs. 20 and 21) all end in one or other of the cerebellar nuclei; none of them pass to the brain stem except through this nuclear relay (Fig. 121).

Efferent tract from the vermis. The Purkinje axons of the vermis end in the nucleus fastigii (tecti) and nucleus globosus, perhaps also in the nucleus emboliformis and the nucleus dentatus. From the nucleus fastigii axons, most of which decussate between the nuclei, pass to the vestibular nuclei. Other fibers come from the nucleus globosus (Fig. 121). Here a

relay is formed by way of the medial longitudinal bundle to the nuclei of the eye muscles and some neck and trunk muscles and by way of the vestibulospinal tract to the rest of the trunk muscles. André Thomas believes that many fibers from these central nuclei of the vermis pass directly to the spinal cord alongside of the vestibulospinal fibers. Thus the vermis through its central nuclei sends fibers to a large number of lower motor neuron cells—probably to all those supplying eye and head-turning muscles, tongue, laryngeal and pharyngeal muscles, to trunk muscles, and to the muscles of the proximal segments at least, of the limbs. It is not certain whether these nuclei of the vermis contribute fibers to the branchium conjunctivum.

The efferent paths from the hemispheres. The axons of the Purkinje cells of the cortex of the cerebellar hemispheres end in the corpus dentatum. The cells of the corpus dentatum by their axons form the brachium conjunctivum (Figs. H and 121). Each brachium conjunctivum decussates with its fellow under cover of the inferior colliculi (Fig. K); this decussation is complete. After this decussation each brachium splits into ascending and descending fibers. The descending fibers can be traced down a short distance only, and nothing more is known about them. The ascending fibers end partly in the red nucleus and partly in the optic thalamus (Figs. L and M). These are entirely crossed connections. It is possible that there is also a thalamocortical path to convey cerebellar influences to the cortex cerebri.

The large cells of the red nucleus form a descending tract in the spinal cord called the rubrospinal tract which immediately decussates with its fellow in Forel's (rubrospinal) decussation (Figs. M and 67). It then descends dorsal to the medial lemniscus in the mesencephalon (Fig. J), medial to the spinal tract of the fifth nerve in the lower pons and medulla oblongata (Figs. I to G), and in the cord it lies ventral to the lateral cerebrospinal tract (Figs. C to A). Its axons end in direct relationship with the cells of the lower motor neurons. This is the chief, and perhaps the only, efferent tract from the cerebellar hemispheres to the lower motor neurons; it may contain efferents from the vermis also. The double crossing—first of the brachium conjunctivum and then of the rubrospinal tract—secures a homolateral influence of the cerebellar hemisphere on the lower motor neuron; the cerebellar influence on the thalamus and cerebral cortex, on the other hand, is crossed. With the exception of median line lesions in the vermis, clinical evidence shows that the cerebellar influence over the lower motor neurons is overwhelmingly homolateral.

The examination of specimens of congenital absence of the cerebellum furnishes striking demonstrations of the nuclei and tracts directly connected with it. A case of congenital absence of the cerebellum recorded by Warrington and Monsarrat, *Brain,* XXV, 1902, showed also absence of temporo-pontine and fronto-pontine fibers, of the pars basilaris pontis, the nuclei pontis and brachia pontis; absence of the central tegmental tract, the inferior olives, the olivocerebellar fibers, the posterior spinocerebellar tracts and the restiform bodies; also there was absence of the dorsal and ventral superficial arcuate fibers, the nuclei of the ventral superficial arcute fibers, and the ventral spinocerebellar tract. The cerebellar nuclei, including the corpora-dentata, the brachia conjunctiva and the red nuclei were absent. Meynert's decussation was well marked, but Forel's decussation was at least doubtful, if one judges from the figures, though the authors describe it as present. It is possible that Forel's decussation in the absence of the cerebellum might form a pallidospinal tract.

SUMMARY OF CEREBELLAR PHYSIOLOGY

The physiology of the cerebellum is a very large and complex subject and only the briefest summary can be given here. It will be found amplified in the section on applied anatomy. Gordon Holmes (*Brain,* 1917) had the opportunity to examine forty cases of gunshot injuries of the cerebellum; being operative cases these compared favorably with laboratory experiments in the limitation of the lesion and the accuracy of its localization, and they had the advantage over animal experiments of coöperation by intelligent patients. In the main Holmes' conclusions confirm those reached by laboratory methods.

Cerebellar functions, as defined by Holmes, are here briefly summarized. Cerebellar lesions produce no defect in consciousness. The cerebellum is not especially an organ of equilibration aside from its intimate relation with the vestibular nuclei. The vermis seems to be closely related to movements requiring the association of bilateral muscles, such as the movements of the head, neck and trunk, and movements required for phonation and articulation; all of these are most seriously affected in lesions of the vermis. Aside from this there is no reason to believe that there is any localization of function in the cerebellum, since Holmes found that limited lesions have effects which, though milder and more evanescent than when the lesions are larger, affect areas of wide distribution. However, neurologists in general differ much on the question of

cerebellar localization. Some claim very definite arm and leg areas in the hemispheres.

The cerebellum is the "head ganglion of the proprioceptive system" (Sherrington); that is, of the whole system concerned in muscle-tendon-joint and vestibular sense. "The cerebellum receives and interpets proprioceptive impulses from all parts of the body, and by virtue of these keeps the motor mechanisms in such a state of tone that they can respond promptly and efficiently to voluntary impulses; and it thus assures the correct coöperation of the separate motor centers that are concerned in individual acts." (Holmes.)

It is probable that the cerebellar nuclei can act largely independently of the cerebellar cortex, which may act mainly as adjuvant to the nuclei. Slowly developing lesions or acute lesions after the lapse of time admit of a great deal of compensation by the cerebral cortex in mammalia, perhaps by the optic lobes in birds.

The chief functions of the cerebellum appear to be (*a*) the maintenance of muscle tone, (*b*) the coördination of muscular action.

Cerebellar symptoms are bilateral if the vermis is affected, homolateral in proportion as the lesion is one-sided. The chief symptoms of cerebellar defect are (*a*) loss of muscle tone, so that the muscles feel soft and flabby; (*b*) asthenia, lack of strength, so that the patient tires easily; (*c*) slowness in the initiation and stoppage of each movement; (*d*) discontinuity and irregularity in the maintenance of muscular contractions, so that muscular action lacks smoothness and becomes jerky; (*e*) cerebellar ataxia; the patient reels like a drunken men and often speaks like one; (*f*) static intention tremor; the hands sway on attempts at fixation; (*g*) nystagmus, especially marked when the patient looks toward the side of his lesion. The eyes show slow asthenic failure, they swing gradually toward the middle line with a quick, jerky return to the lateral position in which the patient desires to fix them. All these symptoms are most marked on the side of the lesion. Symptoms due to a lesion in the vermis tend to be bilateral and are more grave than those due to a lesion in a hemisphere.

Irritative lesions of the cerebellum, as those produced by meningitis, cause cerebellar fits which are characterized by *tonic* spasm of all the muscles of the body; the typical position assumed is that with the legs, trunk and neck in extension, one arm extended and the other slightly flexed. This suggests an exaggeration of the running attitude (Hughlings Jackson). It is, however, more likely that so-called cerebellar fits are due

to cerebellar activity unopposed by supracerebellar control, that is, they are due to loss of cortical and pallidal control over the cerebellum, not due to cerebellar irritation (see decerebrate rigidity, page 290).

A possible *cerebello-thalamo-striate* path completed by an imperfectly known pallidal descending tract with relays in the red nucleus, corpus hypothalamicus or substantia nigra may furnish the anatomical basis for the automatic activities of Goltz's and Luciani's dogs which were deprived of the higher centers in the cerebral cortex (neopallium), yet were capable of walking coördinately, feeding and sleeping. They had lost individuality and become impersonal automata.

THE CORPORA QUADRIGEMINA

While of the utmost importance to the motor mechanism of lower vertebrates (fishes, amphibians, reptiles and birds), yet the corpora quadrigemina appear to be of very minor importance in higher vertebrates, as experiments on cats and monkeys show. Experimental lesions limited to the gray matter of the colliculi cause no visible symptoms except very temporary contraction of the pupils (Ferrier & Turner, *Brain,* XXIV). A little has been said about them in speaking of the optic and cochlear nerves, and more will be said in the applied neurology in treating of mesencephalic lesions.

In man the outstanding symptoms of lesions closely limited to the anterior colliculi is paresis of the vertical movements of the eyes with nystagmus on looking up or down. Lateral movements of the eyes do not suffer.

THE EXTRAPYRAMIDAL MOTOR MECHANISM

The extrapyramidal motor mechanism includes the corpora striata, and the subthalamic nuclei, notably the red nucleus, the nucleus hypothalamicus (of Luys) and the substantia nigra. This motor complex is gradually attracting great attention clinically.

These nuclei are too deeply situated for satisfactory experiment in laboratory animals, but modern methods of examining autopsy brains have made possible the accurate detection of small localized lesions. By such careful post-mortem methods it has been found that clinical symptoms of rigidity, tremors, and involuntary movements are frequently associated with disease of one or other of these nuclei.

Anatomical Summary (see Fig. 87, a, f). The corpus striatum is phylogenetically and physiologically divided into the palæostriatum (globus pallidus) which alone is present in fishes, and the neostriatum

which includes the putamen and caudate nucleus. The neostriatum appears first in reptiles and in these and in birds is the chief source of the highest motor impulses; the motor cortex appears only in mammals and in many of these such as rabbits and goats is wholly or nearly absent. It is to be remembered that a dog deprived of its whole motor cerebral cortex walks about with perfect coördination so long as it has its cerebellum and basal ganglia intact; it has only lost initiative and acquired voluntary movements such as the power to give a paw. The neostriatum which includes the caudate nucleus and putamen is a small-celled nucleus; the axons of its cells end in the globus pallidus. It has a few large cells which belong to the pallidal system. The globus pallidus is chiefly composed of large cells of the efferent type, allied in structure to the cells of the lower motor neurons. It appears to be controlled by the caudate nucleus, putamen and thalamus. Its axons end in the red nucleus, the nucleus of Luys (hypothalamic nucleus), the substantia nigra, the thalamus, and perhaps in other nuclei of the tegmentum mesencephali. S. A. K. Wilson states that the caudate nucleus and putamen receive collaterals from the pyramidal axons.

The tracts which convey pallidal influences to the lower motor mechanism are the rubrospinal tracts, and unknown descending tracts from the nucleus hypothalamicus and the substantia nigra. Clinically they appear to be all crossed.

The physiology of these bodies can be vaguely guessed from clinical symptoms associated with disease limited to each. More will be said of this in Part III. At present it is enough to state that disease of the caudate nucleus and putamen is apparently associated with the appearance of the involuntary movements seen in patients suffering from chorea, athetosis (Figs. 200, 201), and torsion spasms; and that disease of the globus pallidus is characterized by the rigidity, tremors and loss of automatic movements characteristic of paralysis agitans (Parkinson's disease). The influence is crossed if one side only be affected. If both the neostriatum and palæostriatum be involved, rigidity and tremors may be combined with choreo-athetoid movements (Figs. 200, 201, 133 to 135). Disease of the red nucleus is most frequently associated with tremors and cerebellar symptoms (crossed), while disease of the substantia nigra leads to rigidity. Thus the globus pallidus appears to have a controlling action on muscle tone, while the caudate nucleus and putamen regulate reflex automatisms and prevent that continuous overflow of afferent impulses into efferent paths which results in forced involuntary movements. The

relation of the neostriatum to chorea, athetosis, and torsion spasms is still far from settled. In the *Lancet,* Aug. 8, 1925, S. A. K. Wilson reviews the whole subject exhaustively. He regards the striate body as tone controlling, its destruction allowing hypertonicity. It also regulates the whole mechanism of neuro-muscular rhythm; the absence of striatal control allows tremors to occur. Mme. Vogt and most French neurologists incline to the view that the absence of neostriatal control is responsible for choreiform and athetoid movements. A series of cases of torsion spasm following lethargic encephalitis were shown at autopsy to have combined cortical and neostriatal degeneration.

THE FIBRÆ PROPRIÆ OF THE CORD AND BRAIN STEM; THE ANATOMICAL BASIS OF CORD AND BRAIN STEM REFLEXES

A frog with the cord cut just below the medulla oblongata is capable of executing rather complicated movements of a purposeful character and shows considerable muscular tone in the limb and trunk muscles. If the lower part of the mammalian spinal cord be isolated from the upper part and the brain by a section below the phrenic nuclei, after shock has passed off, it can maintain a considerable amount of tone in the muscles connected with it, and the animal reflexly performs purposeful movements in response to cutaneous and other stimuli. These movements are pure cord reflexes and quite unconscious in the ordinary sense of the term, though there has lately been a good deal of discussion of possible cord consciousness. The stimulus may be at some distance from the activated muscles, involving association tracts through many segments. The anatomical basis of this mechanism involves afferent neurons, in most cases association neurons, and sufficient efferent neurons to activate a series of coördinated contracting muscles with simultaneous relaxation of their antagonists.

It has been shown that a considerable number of segments of the cord are linked together by collaterals from the ascending and descending branches of the afferent neurons (Figs. 69, 70, 74, 75 and 76). These occupy the posterior columns. In the anterolateral columns are many ascending and descending association neurons (Figs. 69 and 70) the cells of which are in the anterior and intermediate areas of the gray columns. Many of these association neurons ascend or descend for long distances; the longer fibers always lie nearer the periphery of the cord. After a course of varying length in the white columns, the axons of these association neurons reënter the gray columns and thus link together various segments of the cord for the purpose of intersegmental coördination.

In the brain stem, the medial longitudinal bundle is mainly an association bundle, and is chiefly derived from the vestibular and sixth nuclei. In primitive vertebrates it probably largely subserves the purpose of voluntary movement, as there is no pyramidal tract; but in man it mainly provides for the regulation of eye, head, neck and body movements in response to vestibular indications of the position or movements of the head in space.

THE ASSOCIATION AREAS AND ASSOCIATION AND COMMISSURAL FIBERS OF THE CEREBRAL HEMISPHERES

So far we have been chiefly concerned with primary motor or sensory areas of the cerebral cortex and with fibers to and from these which connect them with lower levels. These are called projection fibers. Study of Figures 142 to 145 shows that while certain limited areas of the cerebral cortex are marked motor or sensory, a much larger field is either labeled as associated with higher motor or sensory memories or is marked "Silent." The clinical term "silent" as applied to an area of the cerebral cortex indicates that hitherto it has been impossible to associate any definite localizing change with experimental stimulation or destruction or with disease of these areas. No animal is so rich in these "silent" areas as man. Presumably they are occupied in the storing up of cell memories either motor or sensory, the linking together of such memories to form abstract ideas, and that association, selection, inhibition and activation of voluntary action, which as a complex whole underlies personality and will. All this requires that every part of the cortex should be linked to every other part and this is done by association neurons (Figs. 149 and 150). They are the axons of lesser pyramidal cells (Fig. 27) and are roughly divided into short association neurons which link neighboring gyri and long association neurons, which link together gyri which are separated from each other by greater distances.

Some of the chief groups of association neurons are indicated in Figure 149. Clinically the most important association neurons are in the neighborhood of the insula and adjoining frontal, temporal, parietal, and occipital areas—on the left side of the brain in right-handed people. Lesions of these association fibers produce some of the more permanent forms of aphasia (Fig.. 105).

The comissural fibers between the hemispheres. The corpus callosum, which first appears in mammalia, but is absent in monotremes and marsupials, is the only commissure which is important clinically. The

corpus callosum unites similar areas of the two hemispheres to each other and perhaps also dissimilar, but functionally related areas. It also contains collaterals from pyramidal fibers to the opposite cortex. All callosal fibers are intercortical (Fig. 150), that is none pass from the cortex of one hemisphere to the internal capsule of the opposite side by way of the corpus callosum.

The *corpus callosum* has been found absent at autopsy in cases in which nothing during life led to a suspicion of abnormality. This means that a child who is born without a corpus callosum, but with no other nervous defect, can get along pretty well. There is reason to believe that in right-handed people the more complicated volitional movements of the left hand are at least initiated by the will by means of the left side of the brain (superior and middle frontal lobe, Figure 142 "higher motor memories," Figure 173) and transmitted to the upper frontal or anterior central area of the cortex of the right hemisphere. It is further probable that visual impressions received by the right calcarine area are transmitted to the left occipital lobe by way of the splenium of the corpus callosum for interpretation and storage as memories (Fig. 105). A similar arrangement is provided for auditory sensations. Thus the corpus callosum provides for harmonized interactions between the cortical cells of the two hemispheres and its development in the mammalia is in direct proportion to the complexity of the cerebral convolutions.

PART III

APPLIED NEUROLOGY

PART III

APPLIED NEUROLOGY

A FEW NECESSARY DEFINITIONS OF TERMS TO BE EMPLOYED

Paresis means slight or incomplete paralysis.

Paralysis means loss of motion or sensation in a living member. Usually when the word is employed, the *motor* loss is most in mind, unless the term be qualified by the adjective *sensory.*

Flaccid Paralysis is paralysis without rigidity of the member. The paralyzed limb falls flail-like if lifted by the physician and then dropped; it lies loosely at the patient's side. The paralyzed leg is an inert mass extended at hip, knee and ankle and turned outward by its weight. The joints offer no resistance to passive flexion. The muscles feel soft and flabby.

Flaccid paralysis occurs in (*a*) the early period of acute upper motor neuron paralysis (cf. hemiplegia); (*b*) always in lower motor neuron paralysis; (*c*) in complete transverse lesions of the cord always during the period of shock, and usually lasting till the death of the patient; (*d*) in upper motor neuron paralysis when intercurrent conditions interfere with spinal reflexes, such as tabes dorsalis and peripheral neuritis, which interrupt the spinal reflex arc, and diabetes and most cachectic conditions, which lower the conductivity of the neurons.

When flaccid paralysis is accompanied by marked wasting of the muscles and the reaction of degeneration, the disease is in the lower motor neuron (Figs. 154, 155, 158, 162, 163 and 165).

Spastic Paralysis means loss of voluntary motion with rigidity of the paralyzed member. It is always due to an upper motor neuron lesion. The most typical cases are hemiplegia after the period of shock has passed and rigidity is established (Figs. 196 and 197) and spastic paraplegia (Figs. 202 to 206). Where the paralysis comes on gradually without initial shock, there is no initial period of flaccidity. In typical hemiplegia both controls over spinal reflexes have been destroyed by hemorrhage into the lentiform nucleus and internal capsule, namely the pyramidal control, and the pallidal control, but the tonic and postural influence from the

vestibular apparatus and cerebellum are uninterrupted. In the spastic paraplegia of lateral sclerosis the *pyramidal* and *pallido-rubrospinal* controlling pathways over tonic reflexes are interrupted on both sides of the cord (Fig. 170) and the vestibulospinal paths carrying vestibular and cerebellar tonic and postural influences are spared.

Paraplegia means paralysis of both sides of the body below the level of a bilateral complete or incomplete lesion of the spinal cord or cauda equina (Figs. 202 to 207).

Hemiparaplegia is a convenient term used to indicate paralysis of one side of the lower half of the body due to a lesion of one lateral half of the spinal cord. Figure 213 is such a case.

Hemiplegia means paralysis of one half of the body due to a lesion of the pyramidal tract above the pyramidal decussation. Thus the paralysis affects the side opposite to the lesion (Figs. 196 and 197).

Crossed Paralysis is the term used when a lesion of the pyramidal tract above the decussation includes the lower motor neuron of a cranial nerve in the lesion. Thus in Figure 183, b, the softening that has destroyed the pyramidal tract and caused upper motor neuron paralysis of the right arm and leg has also destroyed the emerging rootlets of the sixth and seventh nerves with resulting lower motor neuron paralysis of the external rectus of the left eye and of the whole of the left side of the face.

Monoplegia means paralysis of one limb from a lesion in the opposite anterior central convolution limited to the motor area which supplies the affected limb (Fig. 198).

Diplegia is similar to monoplegia, but affects both arms or both legs.

Dysæsthesia and Hypæsthesia mean impaired or diminished sensation.

Anæsthesia is a general term meaning loss of sensation of any or all forms.

Analgesia means loss of the sense of pain.

Thermoanæsthesia means loss of the sense of heat and cold.

Hyperæsthesia means excessive sensitiveness of the skin or of a special sense organ. Frequently the term hyperæsthesia is loosely applied to the disagreeable or excessive reaction obtained in an area of decreased sensitiveness when the stimulus succeeds in breaking through a zone of diminished conduction. Hyperæsthesia may be reflex. Thus in appendicitis or like affections the tenderness to pressure over certain areas of the abdominal wall is due to hyperexcitability of the cord segment connected with the inflamed viscus, brought about by the continuous influx of irritant stimuli from it. An inflamed appendix is not itself tender to pressure and

it is a mistake to think that the appendix is necessarily directly under the tender spot on the abdominal wall.

Hyperalgesia means excessive reaction to painful stimuli.

Paræsthesia means morbid or depraved sensation. Sensations such as numbness, burning, pricking, tingling, crawling, sense of heaviness, appear spontaneously or replace the sensation normally produced by the stimulus applied.

Ataxia means disordered or incoördinated muscular action. Ataxia may be due to (1) a defect in the sensory paths for muscle, tendon, or joint sense, (2) a defect in the cerebellar coördinating mechanism, (3) a defect in the vestibular nerve, its peripheral organ, or its nuclei.

1. *Ataxia due to a defect* in the afferent paths for muscle, tendon, joint sense is the most frequent variety. The difficulty in adjusting voluntary movements is partly caused by the interruption in the stream of those unconscious afferent impulses that normally pass to the lower motor neuron (defect in the spinal reflex arc) and to higher reflex arcs (defect in the vestibular and cerebellar arcs), but defect in conduction of conscious impulses of muscle sense to the thalamus and sensory cortex also helps to cause the ataxia.

The primary lesion may be in the posterior nerve roots as in locomotor ataxia (Figs. 116 and 190), or in the posterior columns, as in the posterior sclerosis of pernicious anemia (Fig. 103). It may be due to disease of the medial lemniscus, when the pyramidal tract escapes injury (Fig. 181). In this case it is a hemiataxia. Also some lesions confined to the optic thalamus or to the parietal cortex cause hemiataxia (Fig. 220).

A peripheral neuritis may cause a certain amount of ataxia combined with other sensory symptoms and with motor paresis. In all cases the patient is much assisted in performing precise movements by the guidance of sight, so that the ataxia is frequently difficult to demonstrate while the patient's eyes are open.

2. *Cerebellar ataxia* (Fig. 128 C to 130) may be due to defect in the cerebellar afferent paths, namely, the ventral or dorsal spinocerebellar tract, the restiform body or the brachium pontis, or to defect in the cerebellar cortex, or to defect in the cerebellar efferent paths (brachium conjunctivum, red nucleus, rubrospinal tract, or vestibulospinal tract). Unless the lesion is bilateral or is in the vermis, cerebellar ataxia is most marked on the side of the lesion. It is not increased by closing the eyes.

3. *Vestibular ataxia* is closely allied to cerebellar ataxia. For its differentiation reference must be made to more extensive text books. The

errors in coördination are readily compensated for, and recovery occurs early.

The ataxic symptoms vary according to the variety of the lesion.

If the lesion be in the afferent paths for muscle, tendon, and joint sense, the ataxia is greatly increased when the patient closes his eyes. If the patient's legs be affected he cannot stand steadily with his heels together and eyes closed, cannot walk a straight line, cannot touch his toe when his eyes are closed. He does not know the position of his legs if he cannot see them (Fig. 118). Having lost his muscle sense he cannot find his way about in the dark even in the familiar surroundings of his own room. With ataxia of the arms he cannot button his collar, and he bungles over all finer movements. He cannot touch his nose or ear with his eyes closed.

In *cerebellar ataxia* the patient walks in a reeling, drunken manner with short steps and feet wide apart due to his difficulty in maintaining his equilibrium (Figs. 129 and 131). He has difficulty in synergizing his muscles and coördinating his movements (Fig. 130). He carries out movements in a jerky manner. He knows his awkwardness and tries to correct it. His ataxia is not increased by closing the eyes.

Apraxia is defined by Barker as inability to make use of the extremities for certain definite combinations of movements necessary for the performance of certain acts, though there is no paralysis of the muscles. I would substitute for "the extremities" in the definition the phrase "certain groups of voluntary muscles," for motor aphasia is an apraxia. The disease is in one or other of the higher motor centers in the left hemisphere (Fig. 142), notably in the prepyramidal area of the frontal cortex or in the supramarginal gyrus, or else in the callosal fibers from one of these centers to the opposite hemisphere. (See Figs. 173, and "Apraxia," p. 283.)

Aphasia is impairment or loss of the power of expression by speech, writing, or signs.

Diaschisis means loss of contact between telodendria and dendrites. It is von Monachow's theory of the temporary paralysis during the period of shock in central nervous lesions.

Synergy is the term used to designate the coöperation of antagonistic muscles in the production of smooth, well-balanced movement. Thus in flexion of the forearm the triceps is relaxed in exact proportion to the contraction of the biceps. If the student will make a fist, he will notice that the wrist is involuntarily dorsiflexed at the same time that the fingers are flexed. Synergy is a function of the cerebellum.

THE CORD IN ITS SEGMENTAL RELATIONS

Though the primary segmental structure of the body is somewhat obscured by the complex development of the limbs and viscera in higher vertebrates, the cord, and still more obscurely the brain stem, are still distinctly segmental organs (Figs. 94 and 95). The segmental relationship of the cord *to sensory skin areas* is simple in the trunk, decidedly obscured in the neck, but very evident in the limbs. Many clinical observations have enabled neurologists to come to a fairly close agreement as to the skin area supplied by each individual posterior spinal nerve root, and this corresponds clinically so closely to a cord segment that it may be used as a means of diagnosing the level of a cord lesion.

The areas shown in Figure 96 are really radicular areas. That is, each corresponds to the sensory distribution of a single posterior (sensory) nerve root. In the case of a cord lesion which involves an entering nerve root as in Figure 109 it is the segment corresponding to the entering nerve root that is the site of the lesion. The segmental level in deep cord lesions such as syringomyelia may be different. This aspect of the subject is too obscure for treatment in the limited space of this text. On the whole it is better to speak of the areas in Figure 96 as radicular areas.

In attempting to determine the level of a cord lesion one must remember that nerve roots bearing pain, heat and cold ascend two to four segments in the same side of the cord before completing their synapses with the second sensory neuron, and that the second sensory neuron then crosses immediately at the level of the synapsis (compare Figs. 96, a, b, c, with 109). In the head and neck the radicular skin areas are a series of triangles (Fig. 96), in the limbs a series of longitudinal skin strips, and in the trunk a series of nearly horizonal circular bands. The radicular sensory areas of distribution overlap, so that unless two to three succeeding nerve roots be affected the sensory loss may be impossible of detection. However, irritation of a single root may define its cutaneous distribution by cutaneous pain or hyperesthesia, or by an herpetic eruption. In the case of a cord lesion, if the lesion be partial and limited to one segment, the sensory tract loss may be the only apparent one (Fig. 104). Under these circumstances there may be no local loss corresponding to the level of the involved segment, but there may be loss of pain, heat and cold below the level of the lesion.

Besides the bearing of segmental skin areas on the level of a posterior root or cord lesion, there are certain skin reflexes of interest to the neurol-

ogist which are definitely segmental in character. Only a few of these need be mentioned here.

Plantar Reflex and the Babinski Phenomenon. Of all the skin reflexes the most interesting is the plantar reflex. Reference to Figure 95 shows that it depends on the integrity of the short reflex arcs in the fifth lumbar and first and second sacral segments. Normally if the skin along the lateral side of the sole of the foot (Fig. 96, e) be gently stroked with a pencil or pricked with a pin, the toes, including the great toe, are flexed. Any lesion, even one of a very insignificant character, affecting the upper motor neuron (pyramidal tract) to the leg alters this response so that stroking the lateral side of the sole produces *extension of the great toe,* sometimes associated with spreading and flexion of the other toes. This is called the Babinski phenomenon, or a *plantar extensor response,* and is of the utmost value in detecting pyramidal lesions, especially in differentiating organic lesions from hysterical paralysis. It is important to note that in children under nine months, and even in some cases up to two years of age, extension of the great toe on plantar stimulation is the normal reflex. This is probably due to the incomplete development of the pyramidal system at this age.

A few other cutaneous reflexes may be mentioned (Fig. 95). The *bulbocavernosus reflex* may be regarded as a skin reflex. If the fingers be placed on the bulb of the penis and the glans penis in the neighborhood of the frenulum be gently squeezed a contraction of the bulbocavernosus is immediately perceptible. The loss of this reflex is a very valuable early sign in tabes dorsalis affecting the sacral nerve roots. Other skin reflexes are the *gluteal reflex,* which is a slight contraction of the gluteal muscles on stroking the buttock (L. 4 and 5, S. 1); the *cremasteric reflex,* elevation of the testicle by the cremaster muscle produced by stroking the medial side of the thigh (L. 1 and 2); the *subumbilical reflex,* tension of the abdominal wall below the umbilicus produced by stroking the skin below the umbilicus; the *supraumbilical reflex,* tension of the abdominal wall from costal margin to umbilicus, caused by stroking the skin above the umbilicus. The *epigastric or submammary reflex* is a retraction of the abdominal wall in the middle line from near the ensiform process as far as the first tendinous inscription of the rectus, caused by stroking the skin beneath the breast. These are important because they are usually *absent* in pyramidal lesions. They are, however, uncertain and are of little value in bilateral lesions. They are of most value in hemiplegia when alteration in the skin reflexes can be tested on the affected side as

compared with the normal side of the same patient. An abdominal skin reflex, present on one side but absent on the paralyzed side, would give corroborative evidence of a pyramidal lesion affecting the trunk, or if equal on both sides would be strong evidence against such a lesion. Absence of the abdominal reflexes in young people, especially if associated with weakness of the abdominal muscles, points strongly to multiple sclerosis (V. Strümpell, E. Müller, Barker). The abdominal reflexes may be temporarily absent in intra-abdominal disease, and in hysterical anæsthesia of the abdominal wall. *Cutaneous reflexes are exaggerated* during the convulsive crises of cerebrospinal meningitis (acute or tuberculous).

Tendon reflexes. In Figure 95 are shown the chief tendon reflexes with the segment involved in each reflex arc. They are obtained by tapping the tendon in question; the muscles of the part should be relaxed and the patient's attention concentrated on some unrelated voluntary act, such as squeezing the hand of the examiner.

A normal tendon jerk requires a complete reflex arc and an uninterrupted pyramidal tract. The jerk may be temporarily absent in fatigue or exhaustion. It is difficult to elicit in some patients without experience on the part of the examiner. It is absent in any interruption of the reflex arc, such as that produced by peripheral neuritis, tabes dorsalis, or posterolateral sclerosis when the posterior sclerosis is marked. It is absent during the period of shock in cerebral hemorrhage and in complete transverse lesions of the cord; it is exaggerated in hemiplegia and in paraplegia when the lesion of the cord is incomplete. Exaggerated tendon jerks form one of the leading symptoms of a pyramidal lesion.

Ankle clonus is clonic contraction of the calf muscles when the ankle is suddenly dorsally flexed by the physician. It can be elicited readily in pyramidal lesions. Occasionally a false clonus can be caused in hysterical subjects and sometimes in health, but this does not occur with complete relaxation of the muscles.

The segmental supply of a few of the more important muscles is also shown in Figure 95. Clinicians have worked out the segmental supply of most of the skeletal muscles, and tables will be found in works on nervous diseases and at the end of this book. Such tables are valuable for the determination of the exact site of transverse lesions of the cord or of local affections of the lower motor neuron, as in inflammation of the anterior gray column (poliomyelitis). It will be seen that most muscles are supplied by two to three segments. Each fascicle of an anterior nerve

root supplies many muscle fibers and is, as it were, a lesser edition of the whole segmental supply (Sherrington).

Figure 94 is introduced on this page of figures for the sake of completeness. We shall afterwards consider the significance of the autonomic centers in cord lesions. Each group of segments marked gives origin to preganglionic fibers passing to the sympathetic ganglia supplying the several viscera.

The segments marked under the heading "referred pain" receive visceral afferent fibers from each organ mentioned. In irritative lesions of each organ a summation of nervous afferent stimuli passes to the corresponding cord segment. Under the influence of these the segment becomes hypersensitive and creates areas of referred pain and local areas of superficial and deep tenderness to pressure with muscular spasm. These skin areas and muscles are within the region of cutaneous and muscular supply of the segment which is connected with the diseased organ. Thus kidney and ureter pain from a calculus may be referred to the peripheral supply of the twelfth thoracic and first lumbar nerves (see Figs. 94 and 96), and there may be reflex retraction of the testicle by the cremaster and spasm of the iliopsoas and loin muscles, with pain and tender spots in the skin areas supplied by these two nerves.

GENERAL CONSIDERATIONS BEARING ON THE INTERPRETATION OF THE SYMPTOMS OF NERVOUS DISEASES

Certain general considerations which especially require emphasis are here summarized.

All tone, all reflexes, all voluntary motion are dependent on a constant stream of sensory impulses from skin, muscles, tendons, and joints (Fig. 100). The neurons through which these enter must be in touch with the lower motor neuron directly (Figs. 71 and 75) or by association neurons (Figs. 74 and 76). *The lower motor neuron is the only efferent pathway from the cord to striped muscle,* so that its interruption means loss of all tone and complete flaccidity with degeneration.

The upper motor neuron is equally dependent upon afferent impulses for its effective action. Its action is suspended or defective in proportion as it is cut off from these. It is probable that the same applies to all other efferent neurons such as cerebellar efferent, vestibular efferent, and pallidal efferent.

Disease of a neuron may be either irritative or destructive. Irritating agents may be poisons or mechanical irritants.

Irritants and the upper motor neuron. Only the cells of the upper motor neuron (cortical cells) are capable of giving rise to contractions under irritation. Such irritation may be localized as by a local meningitis, tumor, or spicule of bone. The symptoms are then caused by cortical-efferent impulses transmitted through the lower motor neuron to a group of muscles or a limb, producing clonic convulsions. *Generalized cortical convulsions* may be produced by a wave-like spread of a local irritation to the whole motor cortex; or by general cortical irritation secondary to increased intraventricular pressure; or by the influence of irritating poisons on the cortical cells, such as the poisons thought to cause some forms of epilepsy, or those of intestinal toxins which may cause convulsions in children. Such generalized convulsions commence with a tonic phase and then become clonic, ending in a period of exhaustion and coma.

Lower motor neuron convulsions are *tonic* in character. They are probably of the nature of a spinal reflex, and not due to direct local irritation of lower motor cells or anterior nerve roots. Thus in appendicitis, the local spasm of the abdominal wall is due to the cumulative action of irritating impulses from the inflamed appendix on the reflex mechanism in the segments of the spinal cord which supply the muscles overlying the inflamed organ. The general spasms of spinal meningitis are probably due to irritation of the afferent nerve roots. The tonic convulsion of strychnin poisoning and of tetanus are believed to be due to diminished resistance at the synaptic junctions in the spinal reflex arcs.

Fibrillar twitchings are occasionally present in peripheral neuritis, but are especially characteristic of slowly progressive disease of the lower motor neuron cells as in progressive spinal muscular atrophy.

Thus, speaking broadly, *clonic convulsions* are usually due to irritation of the pyramidal cells of the motor cortex; *tonic local spasm* or *general tonic convulsions* are usually due to irritation of the reflex cord mechanism. Irritation of the cerebellar cortex may also give rise to tonic convulsions, the so-called cerebellar fits. It is more likely that cerebellar fits are due to cerebellar activity uncontrolled by supracerebellar centers whose control is cut off or temporarily suspended.

Destructive lesions. The action of the upper motor neuron is exerted on the lower motor neuron directly or indirectly and is threefold. The upper motor neuron exercises (*a*) control over spinal reflexes and (*b*) voluntary activating influences over certain groups of muscles; (*c*) at other times the upper motor neuron action may be of an inhibitory character as when it relaxes a flexed limb or inhibits the anal or vesical sphincter.

If we would understand the effect of destruction of the upper motor neuron we must remember that such destruction removes from the field of nervous action one only of the many influences constantly acting on the spinal reflex arc (Fig. 100). The pyramidal system attains its results by using and controlling all the others (Fig. 100). When it is diseased all other influences are to this extent relieved of control. The result is twofold, first, loss of voluntary action, and second, more or less ungoverned action of all the other reflex centers. While disease in the neighborhood of *pyramidal cells* may by irritation give rise to cortical convulsions, disease in the neighborhood of *pyramidal axons,* that is, anywhere near the pyramidal tract at a distance from the pyramidal cells, can cause only paralytic, and not irritative, phenomena. Destructive lesions of the upper motor neuron anywhere in its course, pyramidal cells or axons, cause loss of voluntary motor control with increased muscular reflexes and a mild degree of rigidity, but without muscular degeneration. This is in contrast with lower motor neuron destruction which cuts off all cerebral and spinal efferent influences to the muscles and produces paralysis, loss of reflexes, flaccidity, wasting, electric reaction of degeneration, and histological degeneration of the muscles involved.

THE SENSORY NEURON

LESIONS OF THE SENSORY NEURON

Complete interruption of any sensory neuron is shown by complete loss of all forms of sensation normally passing by that route.

Partial lesion of sensory neurons may cause defective sensory conduction, which is characterized by delayed reaction and a heightened threshold; a stronger stimulus is required to reach consciousness or produce a reaction than under normal conditions. Also the particular sensory impression perceived may be less definite than is that of the opposite side. This is called hypæsthesia.

Paræsthesia. This hypæsthesia may be accompanied by unusual elements of sensation such as tingling, numbness, prickling, coldness, burning or crawling; such, as for example, is the tingling of the leg, accompanying returning sensation in a "sleeping leg," when the conductivity of the sciatic nerve has been temporarily impaired by pressure. These sensations are due to defective or altered conduction of peripheral impressions to the essential organ of the thalamus; the fault is in the lowest sensory neuron.

Hyperæsthesia. Not infrequently when the lowest sensory neuron is

partially interrupted the resulting hypæsthesia may be accompanied by still another result of altered conduction called hyperæsthesia. Here the stimuli reaching the thalamus through the damaged lowest sensory neuron may so react on the essential thalamic organ as to produce very disagreeable sensations. For example, in a peripheral neuritis or syphilitic radiculitis, a pin prick may be delayed in reaching consciousness from the affected area, and many require more pressure with the pin point to produce pain, yet the sense of pain when it does reach consciousness may be unusually disagreeable, as though it had exploded its way through an obstruction. This is called hyperæsthesia. In the posterior radiculitis of early tabes the patient may find the friction of woolen clothing or a rough bath towel unendurable, or may not be able to bear the friction or weight of the bedclothes on the affected limbs. Yet he may be less sensitive to measured stimuli applied to the same skin area.

Intense pain is characteristic of irritation of peripheral sensory end organs, as in neuralgia due to diseased teeth. *Intense and paroxysmal pain* is also found in inflammatory affections of the ganglia of the lowest sensory neurons; this is present in some cases of tic douloureux (neuralgia of the fifth nerve) and in herpes zoster. *Intense paroxysmal pain in tabetic cases* may be due to the spreading of the luetic process to the root ganglia. Where the nerve roots affected contain visceral afferent fibers the paroxysmal attacks of "tabetic crises" may take the form of visceral pain simulating that of gastric or intestinal spasm, gall bladder colic, renal colic, vesical or rectal tenesmus.

Intense paroxysmal pain over all the opposite side of the body is characteristic of some thalamic lesions, and in this instance is probably due to the interruption of cortical control over thalamic reactions.

Among the *milder pains* of posterior radiculitis there are frequently those irregular muscular and articular pains commonly known as *rheumatic.* Many a tebetic patient will give no early history of pain, but if questioned will complain of rheumatism in his legs which is shown by the later developments to have been really nerve root pain due to the early involvement of sacral and lumbar posterior roots in the tabetic process.

When a *cord lesion presses* on or otherwise partially injures the posterolateral (Lissauer's) tract, it affects the spinal portions of the lowest sensory neuron conducting pain and may be accompanied by considerable pain. "Rheumatic" pains are frequently distressing in the early stages of combined sclerosis, and in syringomyelia. It is possible that these pains

are due to early involvement of the lower sensory neuron fibers in the posterolateral columns. I lately came upon the record of a case of pain, continuous, burning, bruising, unlocalizable with slowly waxing and waning exacerbations, involving the area of distribution of the fifth nerve. The patient had a syringomyelic cavity in the substantia gelatinosa of the spinal root of this nerve (*Revue Neurologique,* July, 1922). With these exceptions *partial lesions* of the sensory tracts in the cord and brain stem are seldom associated with marked painful reactions. Therefore, *where pain is a prominent feature in a case of organic disease of the nervous system,* some part of the lowest sensory neuron, or perhaps the gelatinous substance in the cord or in the bulb, or the thalamus should be thought of.

Lesions of the *sensory cortex* do not cause pain; they cause errors in sensory judgments accompanied sometimes in partial lesions by numbness and tingling referred to an arm, leg or side of the face.

Meningeal *inflammation* as in cerebrospinal meningitis gives rise to headache, not to referred pain. Sometimes it produces local tenderness over certain divisions of the fifth nerve.

Intrathecal tumors which press on the spinal cord cause root pains referred to the peripheral distribution of the roots affected. With these may be hyperæsthetic and hypæsthetic phenomena. If the posterior root ganglia be involved the pain may be of a neuralgic character and may be accompanied by an herpetic eruption. There are also, of course, other pressure symptoms of a paralytic character.

The most instructive article on pain in affections of the nervous system with which the writer is familiar is that in Purves Stewart's "Diagnosis of Nervous Diseases."

In interpreting nervous lesions it is well to remember Hughlings Jackson's law "positive symptoms cannot be caused by negative lesions; when present they are *allowed to occur* by the removal of a restraining influence." The application of this law explains the rigidity of the paralyzed side in a capsular hemorrhage as due to the removal of the combined controlling influences of the pyramidal tract and the pallidal efferent system and perhaps other controlling influences. Similarly, the general rigidity in paralysis agitans is due to loss of pallidal control over reflex muscular action.

MUSCLE TONE AND DECEREBRATE RIGIDITY

In man normal muscle tone is the sum of at least *four activating*

and *two restraining* influences. The *activating influences* (see Fig. 100) are:

(*a*) *The spinal reflex mechanism.* Observations, many of them made during the World War, show that in young otherwise healthy patients who had suffered total division of the spinal cord in the thoracic region and who lived long enough for complete recovery from shock, the lower part of the body showed distinct tone with a tendency to general hypertonicity (rigidity) of all the muscles. This spinal tone is marked in laboratory animals after experimental section of the spinal cord in the thoracic region.

(*b*) The *vestibular reflex mechanism.* This was discussed in Part II in the section on the physiology of the vestibular nerve (page 163).

(*c*) The *cerebellar reflex mechanism.* This also was discussed in Part II under the physiology of the cerebellum (page 165).

(*d*) The *cortical tonic influence.* This emanates from the posterior central convolution, depending on the cortical integrity of muscle sense. As this is lost in lesions of the posterior central gyrus there is a resulting hypotonia; however, this may be masked by paralytic rigidity if there be any accompanying injury to the anterior central convolution (Head, *Brain,* 1918).

These last four agencies for stimulating tone act through the spinal reflex arc; without the integrity of this there can be no muscle tone. But the reflex arc need not be complete in each segment, for the preservation of every third afferent nerve root from a limb will suffice for the maintenance of its tone. It is probable that there is a fifth and even a sixth tonic mechanism in the senses of sight and hearing with efferent tracts from the corpora quadrigemina.

The restraining or inhibiting influences, so far as they are known, are two, the *cerebral voluntary motor system* (upper motor neuron), and the *pallidal efferent system.* Perhaps the *thalamo-rubral tract* also exercises an efferent control. These last, the pallidal and the thalamo-rubral, act through the *rubrospinal tract* which crosses in the mesencephalon and probably also through unknown efferent paths from the nucleus hypothalamicus (of Luys) and substantia nigra. Experiments by Weed (*Journal of Physiology,* 1914) strongly suggest a frontal cortical control over cerebellar tone by way of the pontine nuclei.

Hypertonicity. The tone of a limb or group of muscles may be pathologically raised by (*a*) increased excitability of the reflex arc as by a summation of afferent stimuli, or diminished resistance of its synapses caused

by certain poisons, such as strychnine or tetanus toxin, and (*b*) by the removal of one or both controlling influences.

The *loss of pyramidal control* is mainly expressed by increased tendon reflexes. A pure pyramidal lesion is always characterized by increased tendon reflexes for the part paralyzed, with the Babinski phenomenon if the leg be involved; but only mild increase of muscle tone is to be expected in a pure pyramidal lesion.

On the other hand, the removal of the restraining influence of the globus pallidus, as in progressive lenticular degeneration (S. A. K. Wilson), or paralysis agitans, is characterized by markedly increased tone with rigidity of all the muscles, but without loss of voluntary motor power, and without increased tendon reflexes, or the Babinski phenomenon.

Note. Where a pure pyramidal lesion is accompanied by rigidity, the rigidity is due to the unequal or varying control of the pyramidal tract over different groups of muscles. The pyramidal rigidity in the legs is extensor in type and is due to the weakness of the flexors and to the postural action of the cerebellum; the calf muscles are often remarkably soft. In the upper limb the weakness is more marked in the extensors of the elbow and of the fingers, and in the abductors and lateral rotators of the arm. Hence the typical attitudes of paralytic rigidity are adduction of the thighs, extension of the hip, knee and ankle, and adduction and medial rotation of the arm, flexion and semipronation of the forearm and flexion of the wrist and fingers (Figs. 194, 196, 197).

When both pyramidal and lentiform controlling influences are removed, as in a typical capsular lesion or as when both the pyramidal and rubrospinal tracts are involved in lateral sclerosis, the rigidity is still in the position described both for arm and leg; but under these circumstances all control is removed, the rigidity is much more marked, and both groups of muscles partake of the excess in tone and feel firm to the touch.

Posture in spastic paralysis. In order to understand the posture of a patient with ordinary spastic paralysis one must take into account at least two influences that are left active when pyramidal control alone is removed, or when both pyramidal and pallidal controls are removed.

First: The cerebellar tonic influence appears to have a postural element; cerebellar convulsions are characterized by hyperextension of the neck, trunk and lower limbs, adduction and medial rotation of the arm, flexion and pronation of the forearm, and flexion of the wrist and fingers (Hughlings Jackson's running attitude).

Second: Spinal convulsions, such as occur in tetanus due to hyper-

excitability of the spinal cord, are characterized by extension of the neck, trunk, and lower limbs, adduction and medial rotation of the arm, flexion and semipronation of the forearms, and flexion of the wrist and fingers. If the pyramidal and pallidal control be removed on one side, as in a capsular hemorrhage, the uncontrolled action of the cerebellum and spinal cord will produce the characteristic attitude of hemiplegia, namely adduction and medial rotation of the arm, flexion and semipronation of the forearm, flexion of the wrist and fingers, extension and adduction of the thigh, extension of the leg and foot, with a tendency to extension of the toes, and inversion of the foot (Fig. 196).

Lesions involving the pyramidal tract only, such as disease of the motor cortex or subcortical area, and perhaps localized mesencephalic, pontine, and even rare bulbar lesions (Figs. 180, 182, 183, 174) may be associated with very little rigidity, since the pallidal control of the cerebellum and cord is undisturbed. On the other hand, when both pyramidal and pallidal controls suffer interruption (Figs. 168 and 196), as in capsular lesions, there is marked rigidity.

The rigidity of paraplegia (Figs. 202 to 206) as it occurs in lateral sclerosis (Figs. 170 and 206) may be due to the combined affection of the lateral cerebrospinal and rubrospinal tracts (see Fig. 224, b); the destruction of the first removes the pyramidal, and the second, the pallidal control, while the vestibulospinal tract continues to convey cerebellar tonic impulses, and the proprio-spinal reflexes are undisturbed.

Rigidity of the legs in flexion (Fig. 205) occurs only in diffuse spinal lesions where pyramidal and cerebellar tracts are both interrupted and spinal reflexes are alone in ascendency. In the case of advanced lentiform degeneration illustrated in Figure 134 extreme flexion of the thighs is shown. This condition may be due to the exhaustion of the spinal nuclei of the extensors of the legs by prolonged overaction of these in the early part of the disease, or it may be due to exhaustion of the higher centers by the long severe illness. In old cortical or subcortical lesions of the leg areas on both hemispheres there is a tendency to rigidity of the legs in flexion (Fig. 137). Also in diffuse cerebral lesions in old people. The whole subject of rigidity in flexion is far from being satisfactorily explained. Marie and Foix define rigidity in flexion as the defense (mass) reflex rendered permanent which suggests an instructive line of thought.

Reflexes and spasticity in complete cord lesions. In total transverse lesions of the cord there is for the first two to three weeks absolute loss of all reflexes, both visceral and muscular, below the level of the lesion.

Under the usual conditions of civil practice it is extremely rare for patients to recover from this initial condition of spinal shock, as infection of the bladder occurs and bed sores develop with accompanying fever. These prolong the state of shock till death relieves the patient. In the late war, however, it was repeatedly shown that if a young, vigorous man suffered a complete cord lesion by gunshot wound, and bladder sepsis and bedsores were successfully warded off, cord reflexes would return in from sixteen to twenty-one days and gradually become excessively active. Under such circumstances, strong flexions of the legs may occur as the result of slight stimuli, the bladder and bowels may empty themselves automatically, and genital reflexes are troublesome, as erections and often seminal emissions may follow slight unavoidable stimuli to the thighs, glans penis, or neighboring parts. Bed sores, if they have formed during the period of shock, readily heal, the legs become mildly spastic with little predisposition to assume any particular posture. Even after these spinal reflexes are reëstablished, any depression of the general health and especially the supervention of bladder sepsis or bed sores leads to rapid disappearance of the reactions of the cord below the lesion. It is to be noted that the cord above the lesion does not partake at all in the shock.

Contractures. *Early contractures* in paralytic lesions are due to the continuation of the same reactions as those which cause rigidity. *Late contractures* are due to permanent changes with shortening of certain muscles and lengthening of their antagonists and to changes in bones and ligaments consequent upon these.

Cutaneous reflexes, especially the abdominal reflex and cremasteric reflex, are usually abolished in *upper motor neuron paralysis.* No adequate theory has been suggested to explain this.

Shock and diaschisis. Following hemorrhages into the internal capsule the paralysis is flaccid at first, and only after the lapse of one to three months does it become rigid. The initial period of flaccidity appeared to agree almost exactly with the time necessary to establish pyramidal degeneration and led to the theory that rigidity was due to irritation caused by the degenerated tract. In decerebrate dogs and cats, however, rigidity appears about ten minutes after the brain stem is cut behind the thalamus, before degenerative changes have time to develop. Thus it is clearly demonstrated that the rigidity is due not to the degeneration of the tracts but to the removal of a restraining influence higher up. There appears to the writer to be some parallelism between the latent period of flaccidity in human capsular lesions and the period of spinal

shock which appears in man and to a less extent in lower vertebrates after complete division of the spinal cord in, for example, the upper thoracic region. This condition of "shock" affects only the isolated lower segment of the divided cord and is therefore due to its separation from influences originating higher up. In frogs, the period of "shock" with absence of cord reflexes below the lesion is extremely short, only a few minutes in fact; in rabbits it is longer, but is usually limited to half an hour; in dogs it is longer; days may elapse before typical isolation reflexes can be elicited; while in monkeys the period is still longer. In the late war it was observed that young, healthy men who had suffered complete cross lesions of the thoracic cord, if they were carefully guarded from bladder infection and from sepsis from bed sores, showed typical mass reflexes of the severed cord after a period of complete flaccidity lasting sixteen days or more. This stage of flaccidity has been called the period of spinal shock. In experimental animals it is prolonged in proportion as the animal is dependent on its pyramidal system. In the frog, with no pyramidal system, the period of shock hardly exists, and the cord immediately after section is capable of many complex reactions. In the rabbit, with very poorly developed pyramidal system, the period of shock is still short. In the dog and monkey it increases in proportion as the pyramidal system becomes more dominant. In man the period of shock is long and the shock profound and easily continued indefinitely by the depressing effects of sepsis. In fact until the late war few patients with complete transverse lesions of the cord have ever lived long enough to show spinal reflexes arising from the severed portion of the cord. The shock in both man and animals affects only the cord distal to the lesion. Here there is no question of pyramidal degeneration causing the hyperexcitability of the isolated portion of the cord. The periods of flaccidity, both in the cerebral and in the cord lesions, are equally beyond adequate explanation at the present time, but in both cases it is probable that the characteristic reactions are due to cutting off a higher control. After certain controlling influences are removed by nuclear or tract lesions, some time elapses before the remaining possible nervous activities are established. Von Monakow has suggested that this time interval is due to separation of the synapses in the subordinate portion of the nervous system. This he calls diaschisis. Thus the period of flaccidity after a capsular hemorrhage is due to diaschisis in the subordinate motor mechanism; and the period of flaccidity of the lower parts of the trunk and lower limbs after a transverse lesion in the thoracic cord is due to diaschisis in the synapses of the separated

cord. This can only be regarded as a working hypothesis, but to that extent it is helpful.

LESIONS OF SENSORY NERVES AND TRACTS

For full discussion of the anatomical and physiological considerations see Part II, page 134. These matters and the methods of testing the various forms of sensation are summarized at the beginning of the classification of sensory lesions, page 303.

Cutaneous Nerves. Pure cutaneous nerves usually overlap each other so much that any one cutaneous nerve may be cut, accidentally or unavoidably in operating, without discoverable symptoms. Thus division of the superficial ramus of the radial nerve may be without sensory loss, as the dorsal cutaneous nerves of the forearm, the lateral cutaneous nerves of the forearm, medial cutaneous nerve of the forearm, and cutaneous branches of the median and ulnar nerves together completely overlap the rather extensive cutaneous distribution of the radial nerve on the dorsum of the hand, though this complete overlapping is not capable of demonstration by dissection (Fig. 96, e). On the other hand the epicritic loss in injury to the median or ulnar nerve follows its gross anatomical distribution very accurately (Fig. 86). Possibly this has some relation to the development of Paccinian corpuscles on the terminals of these nerves in the fingers, and the great specialization of the fingers as touch organs.

Mixed peripheral nerve. Complete interruption of a peripheral nerve has been fully discussed in Part II, page 148, and need not be considered again. The description there given applies to division of the ulnar nerve, and similar loss is found after division of the median nerve, and possibly of the medial and lateral plantar nerves; but in the case of the ramus superficialis of the radial (O. T. radial) the anæsthetic effect does not always follow gross anatomical limits. Thus if the radial nerve (O. T. musculo-spiral) be divided as it crosses the outside of the humerus there may be no discernable cutaneous sensory loss either epicritic or deep. In one case within the writer's experience anæsthesia to cotton wool and pin prick was limited to a small area over the dorsum of the metacarpo-phalangeal joint of the index finger. In the same case there was loss of pain on deep pressure over the extensor muscles and there was paralysis of all the muscles supplied by the radial nerve after it crosses the lateral border of the humerus; the brachialis which has a double nerve supply escaped. This deep sensory loss was due to the fact that the posterior interosseous or deep branch of the radial by which deep sense travels was

given off below the site of the injury. In all lesions of mixed peripheral nerves the epicritic loss is distributed over a wider area (Fig. 86) than the protopathic loss and the protopathic loss greater than the loss of postural sense, pressure sense, and pressure pain; but as the roots of the limbs are approached the loss of skin pain becomes more nearly coextensive with the loss of light touch, and in nerve root lesions the loss of pain sense is actually greater than the loss of epicritic sense.

Incomplete interruption of sensory nerves. Injuries or tumors or inflammatory conditions causing incomplete interruption of sensory or mixed nerves give rise to a condition of altered conduction with or without local tenderness. Stronger stimuli may be required to pass the obstruction and conduction may be delayed, yet friction of the clothes or pressure over the cutaneous distribution of the nerve may cause intense prickling and burning sensations. In incomplete injuries to nerves there may be spontaneous paroxysms of tearing, crushing pain, increased on movement. Under this form of irritation the cord segments involved may become exceedingly hypersensitive and form a focus for widespread nervous irritability, including excessive reaction to pin prick and other painful stimuli. This excessive reaction is called hyperæsthesia. It usually accompanies hypæsthesia, by which is meant delayed or otherwise defective conduction. Phenomena of this order account for the condition of "painful stump," which follows amputation in which a nerve trunk has been left too long and is exposed to pressure in the cicatrix. The patient suffers from tinglings, burnings and feelings of muscular twitchings and cramps referred to the part of the limb removed. The segments of the cord which give origin to the nerve roots involved become irritable and the least peripheral disturbance becomes unbearable. Section of the nerve trunk well above all cicatricial tissue will stop conduction in such cases and usually will relieve the pain, though not necessarily at once, as the hyperexcitability of the cord segments may continue for some time after connection with the source of trouble has been interrupted.

Peripheral neuritis. In peripheral neuritis there is a period of irritability when hypæsthesia and hyperæsthesia are combined with *local tenderness* over the nerves involved. The symmetrical distribution of the sensory disturbance at the extremities of all limbs is the important feature in diagnosis. The sensory symptoms assume the glove and sock type of distribution, but *shade off* into normal sensation, thus distinguishing them from the hysterical type which stop suddenly (Fig. 209; for motor symptoms in such a case see Fig. 165).

The ventral rami of spinal nerves (Fig. 99) are liable to injury from stab wounds, especially in the neck, from pressure by a bone tumor as they pass through the intervertebral foramina, from the pressure of a cervical rib, etc. If the injury be of an irritative character defective conduction may be associated with pain referred to the hypæsthetic area. This is illustrated by Figure 210, a, b, c, and d. In cases of complete interruption the anæsthetic area is of the root type of distribution (Fig. 96, a, b, and c).

(**Note.** In speaking of the cutaneous sensory areas corresponding to cord segments illustrated in Figures 96, a, b, and c, we shall hereafter often speak of *root* or radicular areas, not *segmental* areas, as this is the more general terminology. Many writers, especially the French, use the term *segmental distribution* of sensory anomalies when they mean the distal, middle, or proximal *joint segments* of a limb.)

As all muscles are supplied by two or more segments of the spinal cord, so all root areas of skin are supplied by at least two consecutive spinal nerves; thus there is no appreciable sensory loss unless two consecutive spinal nerves or posterior nerve roots be involved; but irritation of a single nerve root may cause pain referred to a single root area or paræsthesia or hyperæsthesia on pricking or scratching this area.

If the lesion be incomplete as in Figure 210, defective conduction may be associated with excessive reaction. Thus conduction may be delayed and greater pressure with a pin point be necessary to elicit pain reaction; but when the patient does complain the complaint is excessive as compared with the reaction in healthy skin areas. This may be tested by scratching with a pencil or match across the radicular area; the patient's face will show an expression of distress whenever the hyperæsthetic area is encroached upon.

GANGLIA OF POSTERIOR NERVE ROOTS AND OF THE FIFTH, SEVENTH, AND TENTH CRANIAL NERVES

Head and Campbell (*Brain,* 1900) have shown that herpes zoster is due to an infective inflammation of one or more sensory ganglia. It is characterized by an erythematous eruption accompanied by vesicles, distributed along a cutaneous area corresponding to one or more nerve roots, and preceded, accompanied or followed by neuralgic pains usually of somewhat wider distribution than the eruption. It may be followed by anæsthesia of radicular distribution, or a hypæsthesia to light stimulation may be accompanied by excessive reaction to grosser stimuli. The disease is

usually unilateral, but the ganglia of several adjoining nerve roots may be affected. Rarely there may be accompanying paresis corresponding in distribution to the root areas affected by the pain and rash. Of special interest is herpes zoster of the external auditory meatus accompanied by neuralgia in the meatus and facial paresis. This is due to inflammation of the geniculate ganglion on the facial nerve, and demonstrates the occasional cutaneous distribution of this nerve. It may be accompanied by loss of taste over the area of distribution of the chorda tympani (Ramsay Hunt). Occasionally an herpetic eruption occurs in tabes dorsalis, in spinal tumors, or in spinal tuberculosis, when the posterior root ganglia become involved in the process. In all these cases the distribution of the eruption and of the sensory symptoms follow root areas, and not peripheral nerve areas.

POSTERIOR NERVE ROOTS

Syphilitic radiculitis (Dejerine), Figure 208. Several adjoining nerve roots may be attacked by a syphilitic inflammation confined to the posterior roots between the ganglia and the spinal cord. Sometimes the posterior roots alone are involved and the anterior roots escape, or both anterior and posterior roots may be affected. The disease is usually unilateral and attacks the cervical roots most frequently, though the lumbosacral roots are often affected. All forms of sensation suffer and not pain, heat and cold only, as in the case of syringomyelia. Late in the disease anæsthesia is marked, but the anæsthetic stage is preceded by a stage of partial compression with pain of radicular distribution. The chief symptom is pain of radicular distribution and neuralgic type, continuous, with acute exacerbations which are often violent. The pain may be tearing, crushing, sometimes darting. Also there may be tingling, numbness, sensations of heat and cold. Motor symptoms are less frequent, never occur alone, and usually come later in the disease. They vary in degree from feebleness to paralysis, are of lower motor neuron type and radicular distribution; they may be preceded by cramps. The pain is increased by coughing and sneezing. Figure 208 illustrates such a case of syphilitic radiculitis in the anæsthetic stage. Figure 191 shows the thickening of the pial sheaths of the nerve roots. Contrast the symptoms in Figure 208 with those in Figure 210 and Figure 215.

TABES DORSALIS

Chronic diffuse syphilitic posterior radiculitis—tabes dorsalis. For

the purpose of this discussion tabes dorsalis (locomotor ataxia) may be defined as a slowly progressive syphilitic radiculitis, which affects the posterior nerve roots proximal to the ganglia almost exclusively, occasionally invades the ganglia, and usually, though not always, commences in the lumbosacral region.

The degeneration of the posterior columns of the cord (Fig. 190) is the most prominent lesion seen post-mortem in this disease, but it is always secondary to the root lesion. Figures 114, 115 and 116 show the order of progress of the lesion. Always the posterior nerve roots are affected first, and the posterior columns of the cord secondarily. In certain forms the optic or auditory nerves are invaded early, but these types we shall not consider. It is incorrect to classify tabes dorsalis as a disease of the posterior white columns. The degeneration of these is simply a necessary sequel to the diffuse posterior radiculitis. The relation of the radiculitis to the posterior spinal ganglia is doubtful. Often these are degenerated, but whether primarily or secondarily is a matter for speculation. Except in its relation to pain and occasional concomitant herpes it is unimportant for our present purpose. The relation of tabes dorsalis to general paresis does not here concern us.

From an anatomical point of view tabes dorsalis amounts to a huge natural experiment in slowly progressive destruction of many posterior nerve roots; nearly the whole train of symptoms is just what might be expected from such lesions and may be considered in the light of experimental section of these roots. The effects of the division of the posterior nerve roots to a limb in a laboratory animal are fully discussed in Part II, pages 144 to 146. If the student will read the discussion of this experiment and remember that in tabes dorsalis the destruction of the posterior nerve roots is the result of an inflammatory process of extreme chronicity and irregular progression and is therefore a process in which pain must figure largely in the early stages; and if he will then read the symptomatology of tabes dorsalis in a textbook of nervous diseases he will find that almost all the characteristic symptoms are readily understood. It is necessary also to remember that irregular groups of nerve roots are invaded at different times and that many groups may be in a stage of irritability while others have passed to conditions of more complete destruction. The process extends over ten or twenty years from its inception and is subject to many periods of exacerbation and amelioration. Here only a few leading symptoms can be briefly touched upon.

Heading the list of symptoms to be expected with pain, one may add

to that the effects of experimental division of all the posterior nerve roots to a limb. The list will thus be:

1. *Pain* of radicular distribution, varying with the intensity of the inflammatory process. This as has been said applies to the tabetic inflammatory process only and not to clean section of the nerve roots.
2. *Anæsthesia* of the area supplied for all forms of sensation. In tabetics the anæsthesia is in proportion to the completeness of the root destruction.
3. *Ataxia* of the corresponding limb.
4. *Loss of reflexes,* with loss of muscle tone and marked flaccidity.
5. *Functional,* but not actual, *paralysis* of the muscles.
6. *Atrophy* of the corresponding muscles from disuse but not degeneration.
7. *Trophic lesions* of the skin, and of the bones and joints after slight traumatism.
8. Usually no shock.
9. *Degeneration* in the posterior and posterolateral columns of the cord; chromatolysis in more than half the anterior gray column cells and in all the cells of the nucleus dorsalis for the extent of the nerve lesion. (From Rendle Short, slightly modified.)

Pain. In the early stages of all inflammatory processes involving sensory nerves pain is a prominent symptom, especially when spinal ganglia are invaded. In a very large proportion of cases of tabes dorsalis stabbing pains are the first symptoms complained of by the patient. Coming early they are not at first recognized as tabetic, and are commonly diagnosed as neuralgia, rheumatism, gout, gall-stone colic, and in women ovarian and tubal disease. The early "lightning pains" are described as atrocious. They are lancinating, boring, twisting, burning; usually at first they shoot down the legs, or into the perineum or round the body, less often down the medial border of the arm. Lancinating pain means pain appearing suddenly and characteristically suggesting a stab in that it feels as if it struck vertically to the surface of the limb. By "shooting down the legs" is meant not that the pains appear to travel down the long axis of the limb but that they appear in a succession of stabs at successively lower levels. To the same group of irritative symptoms belong the attacks of gastric pain with uncontrollable vomiting (gastric crises); attacks of diarrhœa, vesical and rectal tenesmus, genital pain, and genital superexcitability.

Anæsthesia. As the invasion of certain groups of nerve roots advances,

areas of anæsthesia appear which are of the nerve root type of distribution. As in all partial nerve lesions defective sensation may be combined with excessively disagreeable reaction to certain forms of stimuli. Thus such a case as is illustrated in Figure 211 may have anæsthetic and hypæsthetic areas associated with other areas where the friction of a bath towel is intolerable. The "corset" sensation described in Figure 211 is due to the hypæsthesia. Patients are often hypersensitive to cold so that they can no longer bear a cold bath.

Genital anæsthesia may lead to impotence and to inability to empty the bladder from loss of genito-urinary afferent impulses; and recto-anal anæsthesia may cause constant difficulty in managing the bowels and occasionally lead to fecal impaction. Figure 212 illustrates an advanced condition of sensory loss. The anæsthetic areas are of great use in detecting early tabes before ataxia makes its appearance, but often careful search is required before they are found. Figure 117 illustrates anæsthesia of the genitalia and other areas. Analgesia and tactile anæsthesia affecting the genitalia is rare in conditions other than tabes dorsalis and lesions of the cauda equina or conus medullaris. It is never present in hysteria.

Reflexes. The tendon reflexes are lost early, especially the patellar and tendo calcaneus reflexes. The bulbocavernosus reflex, the anal reflex and cremasteric reflex are also often lost. This follows as a result of the interruption of the reflex arc. The loss of the pupillary reflex to light is not easily explained.

Ataxia. Ataxia is the motor expression of loss of muscle sense. Normal persons know the position of their limbs, and can use them coördinately and skillfully by virtue of a constant, though unnoticed, subconscious inflow of sensory impulses from muscles, tendons and joints. This is one form of proprioceptive sense, and is dependent on the integrity of sensory nerves from muscles, tendons and joints. Owing to loss of this sense ataxic patients do not know where the affected limbs are unless they can see them. Figure 118 illustrates the inability of such a patient to tell where her leg is when her eyes are closed. Such patients lose their limbs in bed, may leave a leg behind in getting into a car (Starr), and cannot find their way about their bedrooms in the dark. They cannot stand erect with heels together and eyes closed, but reel and fall (Romberg's sign). They cannot walk backward, balance on one leg, or walk on a straight line, and all these defects are aggravated if darkness or closing the eyes leaves them dependent entirely on the muscle-tendon-joint sense complex (postural sense) for guidance. A patient with ataxia of

the arms cannot button his collar or vest or tie his necktie without the aid of a mirror.

Loss of muscle tone and flaccidity. It has been shown that both spinal and cerebellar tonic influences also depend on a constant inflow of proprioceptive impulses; loss of muscle tone therefore follows the loss of muscle-tendon-joint sense. Such patients can spread the legs abnormally and assume attitudes of extreme joint flexion impossible in health. Figure 118 illustrates a degree of hip flexion which could not be produced without discomfort unless the muscles were hypotonic. The patient feels his loss of muscle tone as muscular weakness, and suffers great fatigue on slight exertion. The muscles feel soft and flabby, and later degenerate and atrophy.

Trophic disturbances. These occur most commonly in the shape of perforating ulcer of the foot (Fig. 119) and of great distension of the synovial cavities in the larger joints (Fig. 120). There may be bony new growths round the joints. Such joint affections and ulcers are peculiar in that they run an almost painless course, and this characteristic frequently furnishes a clew to the diagnosis. Rarefying osteitis may be met with in advanced cases, and painless fractures may occur.

Degenerations. The typical degenerations shown in Figure 190 are secondary to the nerve root lesions. They are most obvious of course in the long tracts of the dorsal columns, but the posterolateral columns and the short roots of the posterior columns also suffer. The cells of the nucleus dorsalis and the cerebellar tracts, and the anterior gray column cells are rarely so much affected as to be noticeable.

PRIMARY POSTERIOR SCLEROSIS

Primary posterior sclerosis. A primary sclerosis limited to the posterior columns is rare; it is usually accompanied by sclerosis of the lateral columns, forming a posterolateral or combined sclerosis (Fig. 171). Still, cases of pure primary posterior sclerosis do sometimes occur, and that illustrated in Figure 103, a and b, is particularly instructive when studied in contrast with the secondary posterior sclerosis of locomotor ataxia which has just been considered. The functions of the posterior columns are described in Part II, page 149. The figures, taken from Thompson's article in *Brain,* Vol. XXXIV, show sclerosis limited to the posterior columns; the long fibers from the leg suffer most. Short posterior root fibers almost completely escape. The history of the case is here presented in very condensed form.

Case I. K., aged 53. Subacute gastric disturbance with marked anæmia (Figs. 103, a and b).

Nervous symptoms. He gradually began to lose the use of his fingers. They "became awkward" so that he could not button his vest. His fingers "seemed dead"; he could not feel anything with them, could not hold a cup or anything in his hands. The hands felt like "boxing gloves" and the fingers felt as if swollen. He had feelings in the fingers like electric shocks (tingling?). Later he could not stand properly, his knees "went sideways" when he stood up. He had stinging sensations in the toes, which seemed as if "swollen to the size of flowerpots." He suffered no real pain throughout his illness.

Physical examination of nervous system. Intellect normal; cranial nerves normal. *Motor system*—no muscular wasting, nor loss of power. Much ataxia of the arms, so that he could not touch his nose with his eyes shut nor with eyes shut make his forefingers meet; he could not walk alone, and with assistance his gait was extremely ataxic; he could not stand alone with his feet together if the eyes were closed (Romberg's sign). He had slight stiffness of the legs.

Reflexes. Elbow, wrist, and knee jerk absent, ankle clonus absent; plantar reflex varied, sometimes flexor, sometimes extensor. It will be noted that most short sensory fibers escaped, especially in the lumbar region.

Sensory system. Pain sense was rather increased (hyperalgesia); it was not lost anywhere; sense of heat and cold and tactile sense to light touch and pressure normal.

There was profound loss of the *sense of passive position,* especially affecting the fingers, hand and forearm, less marked for hips, knees and ankle. There was no loss of the sense of passive position of the toe. When the patient's eyes were shut and the physician moved his fingers, hand or forearm, hip, knee or ankle, he could not tell the position in which the member was moved or placed, and could not imitate the movement. On the other hand he knew exactly how his great toes were moved. Many long fibers for the sacral segments of the cord escaped.

Sense of weight was impaired but not lost. With the eyes closed and weights placed in his hand he could not tell that one pound was heavier than one-half pound, but he knew that it was heavier than one-quarter pound.

Localization as determined by naming the spot touched was good, but if he were asked to place his finger on the place touched on the opposite

arm he generally hit a place one or two inches away from the correct one. This was probably due to the loss of sense of position in the arms.

Tactile discrimination was lost. On the palm he could not tell whether he was touched by one or two points of a compass.

Bone sense as tested by tuning-fork vibration was not recorded.

The essential differences between this case and a case of tabes dorsalis are its comparatively acute onset and the absence of nerve root symptoms —no lancinating pains, no areas of anæsthesia.

Figure 192 shows *posterior sclerosis* secondary to an advanced peripheral neuritis due to leprosy. The diagnosis in such a case depends upon the symptoms of a peripheral neuritis with anæsthesia of the extremities gradually shading off (somewhat as in Figure 209), peripheral lower motor neuron paralyses, and the typical granulomatous lesions of leprosy.

COMBINED SCLEROSIS

Combined sclerosis, illustrated in Figure 171, gives rise to a spastic paraplegia owing to involvement of the lateral cerebrospinal tracts, accompanied by ataxia and other evidence of the loss of posterior column conduction. There are no severe lancinating pains as in tabes. In spite of the spasticity, tendon reflexes may not be exaggerated owing to the disease of the posterior columns, which seems to involve the short posterior nerve roots conveying muscle sense.

In Friedreich's ataxia cerebellar symptoms are added to the other symptoms of combined sclerosis. There is no associated anæmia, and the disease begins early in life and runs a more chronic course (Fig. 189). The cerebellar symptoms are the first to appear.

SYRINGOMYELIA

Interruption of the second sensory neurons for pain, heat and cold in their passage through the spinal gray columns or gray commissure of the spinal cord is one of the early results of **syringomyelia.** For the purpose of this discussion syringomyelia may be defined as a slowly progressive growth of neuroglia (Figs. 164, 187 and 188) which begins probably in the central neuroglia, invades the gray matter, and is accompanied by cavity formation. It may occur anywhere from the medulla oblongata to the conus medullaris, but more rarely invades higher parts of the brain stem or ventricular walls; it is most common in the cervical enlargement. The gray commissure is invaded first (Fig. 111); the anterior and posterior gray columns and white commissure later; and the white columns are

pushed aside and invaded late or not at all. The disease begins usually in adult life, and men are more often affected than women. Figures 187, 188 and 164 show typical lesions. (See also Fig. 224, 9 and 9′.)

If the lesions in Figure 187 be compared with the schema of the tracts in Figure 224 it will be at once apparent that the second sensory neurons for pain, heat and cold are early destroyed; either the cell bodies in the posterior horns are affected (Fig. 224, 9, 9′) or the axons as they cross through the gray matter and decussate in the gray or white commissure to join the opposite posterior spinothalamic tracts (Fig. 224, 9, 9′). Usually these soon become bilaterally affected, though the process may commence with the cell bodies of one posterior gray column first and invade the opposite gray column later. Occasionally the posterolateral column is attacked and then the first sensory neurons for pain, heat and cold may suffer. In all these cases the distribution of the anæsthesia is in accordance with the cord segments involved.

A typical case of syringomyelia in the later stages is illustrated in Figures 163 and 216. There is loss of heat and cold (thermoanæsthesia) and loss of pain sense (analgesia) corresponding to the segmental distribution of the seventh and eighth cervical, and first, second, third, and fourth thoracic segments (compare Figs. 215 and 96). Touch, which has a double path in the cord, is involved only slightly or not at all. There is no loss of postural sense, stereognostic sense, or tuning-fork sense, all of which travel up the posterior columns which have evidently escaped. On the other hand, the anterior gray columns have been invaded and there is lower motor neuron paralysis of the muscles of the forearms and hands (Figs. 163 and 216) as well as the back and upper chest muscles (Fig. 163). This is a typical case, but any part of the cord may be invaded and several widely separated segments may be affected at the same time (Figs. 112 and 113). Where the posterolateral column is invaded there may be root symptoms especially involving protopathic nerve endings in this column. These root symptoms consist of darting pains like those of tabes and arthropathies and other trophic lesions. Owing to the early thermoanæsthesia the first thing which the patient may notice is his tendency to burn his fingers painlessly.

Syringobulbia. The floor of the fourth ventricle is subject to an invasion by the central neuroglia which destroys the nerve nuclei in this situation. This may occur independently or as part of a similar lesion in the spinal cord. When the oblongata is affected, there may be lower motor neuron paralysis of the face, tongue, palate, pharynx and larynx;

drooping of the upper eyelid and dilatation of the pupil from destruction of the bulbar control of the pupil dilating center in the upper thoracic cord. Nystagmus and vertigo from invasion of Deiters' nucleus are among the possible symptoms. There is a special tendency for the disease to spread in the neighborhood of the roots of the glossopharyngeal nerve affecting the sense of taste on that side of the tongue. As the process spreads it may cut off the deep arcuate fibers as they pass from the nucleus gracilis and cuneatus, and thus interrupt the second sensory neuron for muscle-tendon-joint sense on the same side of the body as the lesion and lead to degeneration of the opposite medial lemniscus. If it invades the spinal tract of the fifth nerve and its gelatinous substance there may be trigeminal neuralgia and homolateral loss of pain, heat and cold in the face and mucous membranes supplied by the fifth nerve.

The **posterior spinothalamic tract and its lesions** are sufficiently discussed in Part II, page 151.

The *anterior spinothalamic tract* (Fig. 69) appears in most cases to be an alternate route for simple touch and pressure. As Figure 69 shows it is a crossed route; the crossing takes place 2 to 6 cm. above the segment where the posterior nerve roots conveying these forms of sensation enter. The other possible route for touch and pressure is in the homolateral posterior column. Special interest attaches to these alternate routes in lesions limited to one lateral half of the spinal cord (Brown Sequard paralysis), and this tract will be discussed under this heading.

SENSORY TRACTS IN THE MEDULLA OBLONGATA AND PONS

In considering lesions involving sensory tracts in the medulla oblongata and pons, it is necessary to have constantly before one a vivid picture of the nuclei and tracts at the level of each supposed lesion and to have accurate knowledge of their interrelationship. These should be reviewed with the help of Figures E to I in the long diagram.

The lower sensory neurons for the sense of active motion, weight, and passive position—the muscle-tendon-joint sense complex—for compass sense tactile discrimination), and vibratory or bone sense, and in some cases at least for touch and pressure travel upward homolaterally in the posterior columns. In the medulla oblongata these neurons are relayed by second sensory neurons whose cell bodies are in the nucleus gracilis and nucleus cuneatus, and whose axons cross as deep arcuate fibers to the opposite medial lemniscus (Figs. E and F). At this level tactile discrimination branches off from the other forms of sensation which it has hitherto

accompanied to take some undetermined separate path in the formatio reticularis (Holmes, *Lancet,* 1912, Vol. I). In the medulla oblongata and pons the medial lemniscus carries the sense of active and passive motion and passive position and vibratory sense for the opposite side of the body, but it no longer carries compass sense. In the pons the medial lemniscus is joined by arcuate fibers from the lateral sensory nucleus of the trigeminal nerve which convey the same sense complex, but not pain, heat and cold, for the sensory distribution of the fifth nerve, and for the seventh nerve in so far as that contains fibers for pressure and muscle sense.

The posterior spinothalamic tracts, in the medulla oblongata and lower part of the pons, lie ventromedial to the spinal tract of the trigeminal nerve; higher up in the pons they lie just dorsolateral to the lateral border of the medial lemniscus. They join the medial lemniscus in the mesencephalon. The position of the anterior spinothalamic tracts in the medulla oblongata and pons is not well understood.

The trigeminothalamic tract is formed by the second sensory neurons for pain, heat and cold for the trigeminal nerve. Its cell bodies are in the gelatinous substance associated with the spinal tract of this nerve (Figs. E to H). The axons cross as arcuate fibers and form a separate tract in the medulla oblongata imbedded in the reticularis grisea between the gelatinous substance and the medial lemniscus. The tract is separate from the spinothalamic tract so that clinically the spinothalamic tract may be involved in a lesion in the medulla oblongata, while the trigeminothalamic tract escapes. In the pons these tracts come very close together and they all join in the mesencephalon.

In addition to these sensory tracts certain of the efferent tracts in the oblongata and pons are of clinical importance. *The pyramidal tract* lies on the ventral surface of the oblongata and is imbedded in the basilar portion of the pons. If it is involved in the lesion, the result will be an upper motor neuron paralysis, of a pure pyramidal type, affecting the opposite side of the trunk and limbs.

The rubrospinal tract (Figs. H to E) lies near the lateral border of the medial lemniscus in the upper pons and medial to the spinal tract of the trigeminal nerve in the lower pons and oblongata. It is the chief efferent tract from the cerebellar hemispheres, and is relayed from the opposite red nucleus. It is also at least one of the efferent tracts from the opposite globus pallidus, though perhaps not the only one. It is homolateral in distribution from the mesencephalon down, as the crossing

takes place between the red nuclei (Fig. L). Its involvement in a lesion may be expected to produce cerebellar tremors and dyssynergia on the same side.

The medial longitudinal bundle carries two sets of fibers of clinical importance.

First, the medial longitudinal bundle carries fibers from cells of association neurons in the homolateral sixth nucleus, which provide for the medial rotation of the opposite eye in association with lateral rotation of the eye on the side of the lesion (see Fig. 178). In the case illustrated the involvement of the emerging sixth nerve (nerve to the external rectus of the eyeball) has caused lower motor neuron paralysis of the left external rectus with inability to turn the homolateral (left) eye outward. The involvement of the left medial longitudinal bundle has cut off fibers from association neuron cells in the left sixth nucleus, that go by way of this bundle to the third nucleus to provide for the medial rotation of the right eye, that is for turning it to the left side in conjugate deviation of the eyes to the left. It is uncertain whether this medial rotation of the eye in coördination with the lateral rotation of the opposite eye is provided for by decussating fibers from the opposite third nucleus or by homolateral fibers. In the first case the association fibers in the medial longitudinal bundle would end in the homolateral third nucleus; in the second case they would cross in the immediate neighborhood of the third nucleus to the side opposite from their nucleus of origin.

Secondly, the medial longitudinal bundles carry fibers from Deiters' nucleus which provide for the turning of the eyes toward the same side as the nucleus in question. The eye symptoms in a lesion of the left Deiters' nucleus are explained with Figure 179; both eyes are weakened for conjugate movements to the left.

Deiters' nucleus and the vestibulospinal tract. Deiters' nucleus in the lateral bulbopontine angle of the floor of the fourth ventricle (Fig. G) and the vestibulospinal tract in the lower pons and bulb (Figs. G to E) merit attention in bulbopontine and bulbar lesions. Besides exercising the vestibular influences on the eyes which have just been considered, Deiters' nucleus by way of the vestibulospinal tract conveys homolateral cerebellar and vestibular tonic and coördinating influences to the lower motor neurons. In the lower pons and oblongata the rubrospinal and vestibulospinal tracts are so close together that they can scarcely be involved separately in a local lesion. Therefore homolateral cerebellar and ves-

tibular symptoms—asthænia, atonia, tremors, asynergia, dysmetria, adiadokokinesis—should be carefully looked for in all lesions of the formatio reticularis in the lower pons and medulla oblongata.

The restiform body is an important afferent tract to the cerebellum. It is situated dorsolaterally in the upper part of the medulla oblongata and is to be taken into account in all lesions in this neighborhood. Destruction of the restiform body gives rise to well-marked homolateral cerebellar symptoms (Fig. 175, 129).

The lower motor and sensory nuclei and the nerve roots of the cranial nerves from the fifth to the twelfth must be considered in all lesions in this region. Their involvement causes homolateral symptoms of the lower motor neuron and the lowest sensory neuron type (Figs. 174, etc.).

Parasympathetic nuclei and autonomic control in the medulla oblongata and lower pons. The salivary nuclei of the facial and glossopharyngeal nerves are situated in the bulbo-pontine region. The dorsal afferent and efferent nuclei of the vagus represent vagotonic influences over the heart, lungs, and upper gastrointestinal tract. Somewhere in the formatio reticularis of the oblongata is a *nucleus whose stimulation causes glycosuria.* The *vasomotor mechanism and sweat secretion* are controlled homolaterally by a center in the medulla oblongata.

In the reticularis grisea of the medulla oblongata there is a nucleus which controls the pupil dilating center in the eighth cervical and first and second thoracic segments of the spinal cord (Fig. 94) and the center innervating the involuntary muscles of Mueller in the same segments. This nucleus sends fibers homolaterally down the cord to these segments, and destruction of it may cause homolateral contraction of the pupil, slight drooping of the eyelid and recession of the eyeball.

If all these facts be borne in mind the interpretation of such lesions as are illustrated in Figures 174 to 179 is an instructive exercise in applied anatomy. The legend attached to each figure carries sufficient explanation. Patients with this type of lesion are usually too ill to permit of careful search for cerebellar symptoms.

Mesencephalic sensory and mixed lesions. In the mesencephalon the medial lemniscus carries all forms of sensation from the opposite side of the body including the face; its destruction here causes heterolateral hemianæsthesia for all forms of sensation. Nuclear or root symptoms of the homolateral oculomotor nerve are usually present. Homolateral or heterolateral cerebellar symptoms may be observed, depending upon

whether the brachium conjunctivum or the red nucleus is included in the lesion. If the basis mesencephali be involved there is crossed hemiplegia; this should be of the pure pyramidal type as regards spasticity. The upper face, masticatory muscles and tongue are less affected owing to the influence of homolateral corticonuclear fibers in the opposite side of the mesencephalon (Fig. 181).

As has been said in speaking of the physiology of the *corpora quadrigemina* the characteristic symptom of involvement of the superior colliculi is paralysis of the upward and downward movements of the eyes. The presumption is that the lateral movements of the eyes are controlled from other centers than the superior colliculi, probably the corpora dentata cerebelli, as well as certain cortical cerebral centers.

While involvement of one *medial lemniscus* in the mesencephalon and hypothalamic region usually causes loss of all forms of sensation in the opposite side of the body, there are rare cases where even here certain forms of sensation may escape.

The *aqueductus cerebri* is of the utmost clinical importance in tumors of the mesencephalon, especially in those of its tegmental portion. Pressure on the aqueduct produces increased pressure in the lateral and third ventricles, and this causes severe headache, vomiting, and the early development of choked disc. There may possibly be tonic convulsions with general rigidity, both of the decerebrate type; the head is hyperextended, the trunk and limbs extended, and the forearms hyperpronated so that the palms are turned outward.

SENSORY LESIONS OF THE SUBTHALAMIC REGION

Reference to Figure M shows that the medial lemniscus in the subthalamic region is in very close proximity to the red nucleus, the nucleus hypothalamicus, the striorubral radiation, the basis pedunculi, and the ventral nucleus of the thalamus. The optic tract is in close contact with the basis pedunculi. Here all forms of bodily sense for the opposite side of the body, including the area supplied by the trigeminal nerve, are so closely grouped in the medial lemniscus that with rare exceptions the most limited lesion of one medial lemniscus causes loss of all forms of bodily sense for the opposite side of the body.

Simultaneous involvement of neighboring structures should cause symptoms as follows:

Red nucleus. This is an intermediate station in the cerebello-rubrospinal tract, and the striorubrospinal tract (see cerebellum and corpus

striatum). Its involvement should cause heterolateral cerebellar symptoms—asynergia, dysmetria, ataxia, atonia, asthænia, and termor.

Basis pedunculi. In proportion as the pyramidal tract suffers there would be heterolateral hemiplegia, including upper motor neuron paralysis of all the opposite cranial nerves.

Optic tract. Should the optic tract suffer there would be homonymous hemianopsia for the homolateral half of each retina, that is, for the opposite side of each visual field with Wernicke's oculopupillary sign.

The *olfactory mechanism* is too much out of the way to suffer.

The *auditory sense* has bilateral representation and therefore would escape. The proximity of the third and fourth nerves must be remembered.

LESIONS OF THE THALAMUS

The effects of lesions of the thalamus and especially the manner in which the various modes of sensation are affected are summarized in the classification in the appendix, to which the student is referred.

If one includes the geniculate bodies or metathalamus with the thalamus proper all the secondary sensory neurons end in one or other of the thalamic nuclei, with the possible exception of the secondary vestibular path to the cerebrum whose thalamic connections are unknown.

The medial geniculate body (Fig. L) is the subcortical center for hearing. In it ends the lateral lemniscus, which is the secondary auditory path from the cochlear nuclei of both sides, and from it springs the auditory radiation to the superior temporal gyrus (Figs. L, 90 and 92). The lateral lemniscus probably also sends short efferent neurons to the inferior colliculi. It is possible that indefinite general impressions of sound reach consciousness in this nucleus. It is known that cats in which the brain stem has been sectioned above the inferior colliculi are exceedingly sensitive to sound (Bazeth, *Brain*, 1920), but this is probably a direct reflex through collaterals from the lateral lemniscus to the colliculi. A lesion of the medial geniculate body does not cause deafness of either ear because each ear has bilateral cerebral connections.

Lateral geniculate body and pulvinar. These nuclei are end-stations for the secondary optic neurons (Figs. L and 105). They contain cells whose axons form the tertiary path for sight. These reach the area striata surrounding the calcarine fissure by way of the *optic radiation* (Figs. 87, b, 90, 93 and 105). Destruction of either of these nuclei or interruption of the optic radiation causes homonymous hemianopsia of the same half of each retina or the opposite half of each field of vision with normal oculo-

pupillary reaction (see Fig. 105). It is doubtful whether destruction of the pulvinar alone causes hemianopsia if the optic radiation escapes.

The anterior nucleus of the thalamus (Figs. 87 and M) is regarded as a subcortical end-station for the olfactory paths, which appear to reach it through the mammillo-thalamic bundle. So far this nucleus has not attained clinical significance.

Lateral and medial thalamic nuclei (Figs. 87 and M). There are left for consideration the lateral and medial thalamic nuclei. They are particularly important, as in them all forms of bodily sense are relayed on their way to the sensory cortex. In the subthalamic region the medial lemniscus has been joined by the spinal thalamic and trigeminothalamic tracts; so augmented, it ends in the ventral part of the lateral nucleus of the thalamus. In this situation, therefore, the various forms of bodily sense are still somewhat massed together. There is clinical evidence that a redistribution and segregation now takes place by short neurons to the dorsal part of the lateral thalamic nucleus. Thus touch, pressure, pain, heat, and cold, and the sense of spacial relations in one, two, and three dimensions have each separate representation in the complex cell groups which constitute the dorsal part of the lateral thalamic nucleus. From this nucleus two distinct relays take place:

1. *Relay to the essential organ of the thalamus.* The fundamental senses of touch, and especially touch as felt by the hairs, as well as pain, heat and cold, are relayed to the medial thalamic nucleus which probably represents Head and Holmes' essential organ of the thalamus. Here they undoubtedly reach consciousness, and are associated with feeling tone reactions, that is, with a conscious sense of comfort or discomfort, pleasure or pain. This nucleus has no direct cortical connection, but it may be relayed to the cortex for the purpose of storing pleasurable or painful memories and thus establishing voluntary reactions; this relay probably goes to the frontal lobe. It is also connected by nonmyelinated neurons with the caudate and perhaps with the lentiform nucleus for subcortical motor reflexes. Further, the essential organ of the thalamus has an indirect or corticothalamic connection from a wide area of cerebral cortex to the dorsal part of the lateral thalamic nucleus; thus thalamic reactions are brought under cortical control. If this control be removed physically or psychically, thalamic reactions are apt to be excessive.

2. *Relay from the dorsolateral thalamic nucleus to the parietal cortex.* The "crude awareness" of touch, pain, heat and cold, and their association with the quality of comfort or discomfort, pleasure or pain, is a function

of the essential organ of the thalamus. On the other hand, all spacial relations, all correct reference of points of contact to the regions of the body touched, all comparison of degrees of temperature, of pressure, and of pain, and the relation of these to past experiences of a like nature, all storing of fresh sensory experiences as cell memories (if one may use this expression for want of a better), are the function of the cerebral cortex. Also all judgments of the position of the limbs or body in space are based on sensory "schemata," to use Head's expression, of past spacial relations as conveyed to and registered in the parietal cortex. Differences in weight too can be judged only through the agency of the cortex, whether they be estimated by the varying pressure exerted by differing weights of like extent and form, or by actively balancing objects of similar size and form as one usually compares weights in the hand.

Judgments of space in one dimension are represented in the cortex by the power to identify the spot touched. Judgments of space in two dimensions are readily compared by the ability to recognize two points of a compass as two when they touch the skin simultaneously at measured distances. On spatial judgments depend the recognition of the shape of flat objects. The tactile recognition of the form and size of solid objects depends on very complex combinations of impressions of one and two dimensional character with those of postural sense involved in the muscular action necessary to grasp the solid body; these constitute judgments of three dimensional space. One can only judge of the shape of solid objects by comparison of present sensory impressions with past experiences; all this is a function of the cerebral cortex in the parietal lobe. The difference in crude sensory perception and sensory judgments may be used to interpret the symptoms of thalamic and suprathalamic lesions.

THALAMIC LESIONS

A massive hemorrhage into the thalamic substance is incompatible with life and the patient never recovers from the shock; accurate diagnosis beyond that of severe apoplectic seizure is not possible. The proximity of the thalamus to the lateral and the third ventricles may permit of hemorrhage into them; under these circumstances general convulsions and early rigidity are likely to be prominent symptoms. The typical rigidity produced by increased intraventricular pressure is extensor in type; the arms and legs are fully extended; the forearms extended and excessively pronated so that the palms look outward; the fingers are flexed. The convulsive seizures are periodic tonic exaggerations of the rigid position;

they are not clonic like the cerebral convulsions of cortical origin (see Wilson, "Decerebrate Rigidity in Man," *Brain*, 1920).

Ventrolateral nucleus. Hemorrhage into the ventrolateral nucleus may not be distinguishable from a similar lesion of the subthalamic region involving the medial lemniscus, because the tracts for bodily sense are still very much massed together.

Dorsal part of the lateral nucleus. In the dorsal part of the lateral nucleus the various modes of bodily sense are grouped with sufficient distinctness that a limited lesion such as softening or a slowly growing tumor may cause sensory loss which varies greatly in different cases. Those simple forms of sensation that make a direct appeal to the essential thalamic organ suffer the least in actual conduction, but the nature of their thalamic appeal is likely to be altered. Thus touch, tickling as produced by lightly brushing the hairs, roughness as elicited by sponging or by drying with a bath towel, pain, heat and cold, appear to pass with difficulty through the thalamus to the medial nucleus, so that somewhat greater stimuli are needed to reach consciousness, but when they do "break through" they seem to reach the essential thalamic organ with explosive force. Thus the thalamic response is excessive and is attended with unusual discomfort. This excessive response may be due to interruption of cortical control over thalamic reactions. Any one or several of the various forms of sensation may be entirely lost, while others suffer little or not at all. The sense of posture and of space in its three dimensions (spot finding, compass sense, tactile recognition of size and form) suffer most, probably because their more elaborate differentiations require more complex thalamic connections.

Much clinical experience is necessary to differentiate thalamic from cortical lesions by the minutiæ of sensory loss. In thalamic lesions the sensory loss is distributed all over the opposite side of the body; in cortical sensory lesions as in cortical motor lesions the loss is likely to have a much more limited distribution. Further, as those parts of the body which are most highly endowed with discriminating sense are most highly educated, have the greatest cortical representation, so the hands and feet suffer most in cortical lesions, while the sensory loss in thalamic lesions is more generalized. The excessive response to stimuli and "thalamic pain" are the distinguishing characteristics of affections of the thalamus (compare Figures 220 and 221).

The thalamic syndrome. Dejerine and his pupils by the study of patients with thalamic lesions later confirmed by autopsies have been

able to put together a group of symptoms which in association with each other are characteristic of certain cases of limited lesions of the lateral nucleus of the thalamus. These observations were confirmed by Head and Holmes (*Brain,* 1911, 1912), who added to Dejerine's group the last symptoms in the list given below. Usually all the symptoms are of wide distribution and they are always referred to the side of the body opposite to the lesion.

While wide distribution of the pain and anæsthesia over the whole of the opposite side of the body is characteristic of thalamic lesions, there are occasional cases of thalamic pain and anæsthesia affecting one limb only. These cases are commonest in the thalamic type of lethargic encephalitis (*Revue Neurologique,* June, 1920). The typical syndrome comprises:

(*a*) Persistent loss varying in intensity of superficial sensation, touch, pain, heat and cold for the opposite side of the body.

(*b*) A greater loss of the sense of posture, size, shape and form in three dimensions.

(*c*) Slight hemiataxia and more or less complete astereognosis.

(*d*) Intense pains, "persistent, paroxysmal, yielding to no analgesic."

(*e*) Slight hemiplegia without contractures and passing off rapidly.

(*f*) Choreiform and athetoid movements (probably a neighborhood symptom due to involvement of the neostriatum).

(*g*) A tendency to react excessively to unpleasant stimuli.

Sufficient has been said to make all these intelligible except the presence of choreiform and athetoid movements.

Choreiform movements are irregular movements of an unexpected jerky type which cannot be arrested by the will for any length of time. They are much increased by attention, by excitement, or by any effort to control them or to use the muscles involved. There are often grotesque facial grimaces, especially when the patient is talking. They cease during sleep. *Athetoid movements* are continuous, involuntary, purposeless, twisting movements of the hands or feet and sometimes of the face (Figs. 200 and 201). Many explanations have been suggested to account for these movements; the present tendency is to associate them with lesions of the neostriatum. In thalamic lesions they may be due to the vitiated transmission of sensory impulses through the damaged organ to the corpus striatum, where irregular reaction of the subcortical motor mechanism gives rise to these involuntary movements. (For thalamic case see Fig. 220.)

LESIONS OF THE SENSORY CORTEX

(See Classification, Appendix)

The article by Head, published in *Brain,* 1918, on "Sensation and the Cerebral Cortex," is founded on war injuries of the parietal lobe; it may be regarded as representing the latest ideas on the sensory function of the cortex. The subject is exceedingly complex and only the most general summary can be attempted here.

1. *Nature of cortical activity in relation to body sense.* While all forms of bodily sensation are massed very closely together as they enter the ventral part of the lateral thalamic nucleus, they are immediately segregated in separate cell groups. Probably this occurs in the dorsal part of the lateral nucleus of the thalamus, for here limited lesions may cause the loss of one form of sensation while other forms remain more or less intact. Further, the crude "awareness" of the elementary sensations of touch, pain, heat, and cold reach consciousness in the thalamus and are there associated with the quality of comfort or discomfort, pleasure or pain. The feeling tone must be physical; the psychic element probably depends on thalamo-frontal and thalamo-parietal, or perhaps thalamo-parieto-frontal connections.

Clinical evidence is rapidly accumulating which shows that the cerebral cortex supplies the mechanism which underlies psychic relations to sensation. For lack of better terms we may say that they form the physical mechanism for the storing or registration of sensory memories, the grouping or association of these into sensory "schemata," the comparison (usually subconscious) of present sensations with previous experiences, and the final expression of these complexes in the shape of voluntary actions or inhibitions.

Therefore, in order to investigate the effect of injuries of the sensory cortex, one tries to estimate the patient's capacity for simple sensory judgments. Instead of testing simple touch one tests the power of localization (spot finding, topognosis), the recognition of distances between compass points on the two sides of the body, and the recognition of the posture of limbs, limb segments or digits. Thus one applies tests in the recognition of spatial relations, in one, two, and three dimensional space and estimates the patient's discrimination of quantitative sensory appeal by his judgments of the shape and size of flat objects, the comparative weight of objects similar in size and form, the comparative roughness or smoothness by which he recognizes the texture of silk, cotton or woolen

fabrics, the judgments of differences in graded temperatures of hot and cold test tubes. More complex sensory judgments may be tested by the tactile recognition of familiar solid objects, or the active weighing of objects identical in size and form but differing in weight.

Horsley found that limited lesions of the central gyri are associated with sensory loss which is most marked in the distal segments of the limb (Fig. 221). The sense of passive position of the toes and ankles, fingers and wrist are most interfered with. Touch localization (topognosis) is associated with proximal and pre- or post-axial errors. The compass test shows that great widening of the distance between the points of the compass is needed before they are perceived separately. In all these tests similar areas on the two sides of the body must be compared (Fig. 222).

2. *Anatomical considerations.* According to Head the sensory cortex includes the anterior and posterior central gyri with the paracentral lobule on the medial surface of the hemisphere, the superior parietal, supramarginal, and angular gyri. The anterior central gyrus is preëminently motor; but a strictly localized lesion of the anterior central gyrus is always accompanied by considerable loss of postural sense in the paralyzed member. On the other hand the parietal cortex is preëminently sensory, but any considerable lesion of the posterior central gyrus is accompanied by hypotonia and ataxia, in proportion to the loss of the postural sense and affecting the member chiefly involved in the sensory loss.

Localization of the different forms of sensation in the parietal cortex. Brodman, Campbell, and Elliott Smith agree sufficiently closely when they divide the posterior central gyrus into successive strips of differing microscopic structure (see Figs. 140 and 141), while the immediate neighborhood of the horizontal limb of the inferior parietal sulcus, the lower end of the posterior central sulcus, the superior parietal, supramarginal and angular gyri all show differences in cell and fiber distribution. This of course suggests variations in function.

Flechsig's figures of the order of myelination of these cortical areas are especially suggestive. The posterior central gyrus myelinates early, at the same time as the anterior central gyrus; the superior parietal and lower part of the supramarginal gyri myelinate much later; and the upper part of the supramarginal and angular gyri, last of all. This suggests that the posterior central gyrus is intimately connected in its sensory relations with voluntary purposive movements, and that the areas last

myelinated are associated with sensory syntheses involving considerable mental development. Now Head concludes from observations on actual cases that those sensory functions which seem most necessary to the carrying out of well-directed purposive muscular activities are localized in the posterior central gyrus; these are the varieties of spatial recognition, especially postural recognition (Fig. 142). Judgments of more-or-lessness in touch, heat and cold, and roughness are apparently localized in the parts just above the lateral sulcus—the lower end of the central gyri and the lower part of the supramarginal and angular gyri. Cases are recorded by Starr and others that seem to support the view that the complex sensory syntheses and memory pictures contributing to tactile recognition of solid objects, stereognosis, may be associated with the upper part of the supramarginal gyrus (Fig. 142).

3. *Sensory representation of parts of the body in the cerebral cortex.* It has been shown that the tongue, face, and larynx, each finger, the forearm, arm and shoulder, the trunk, hips, leg, and toes, and the perineal muscles have fairly distinct areas of chief representation in the motor area of the cerebral cortex; but it is not possible to map out similar distinct limitations in the sensory cortex. The parts of the body most highly educated as sensory organs have the widest cortical representation, and the most complex sensory syntheses show the greatest loss in limited lesions of the sensory cortex. The hand and foot, especially the hand, are the most highly educated in bodily sense; the joints of the fingers, the wrist, elbow and shoulder are in the order given most highly trained as organs of postural sense.

Clinically Head finds that in limited lesions of the sensory cortex the hand, and more especially the fingers, suffers most, and the foot next; and that only in extensive lesions is there any marked sensory loss at the elbow and shoulder. In the posterior central gyrus spatial sense in one or other of its forms suffers most in the foot when the upper part of the gyrus is involved. Sensory loss in the foot is likely to be associated with some loss of spatial sense in the little finger, while damage to spatial sense in the thumb is often associated with some sensory or motor loss in the tongue or face, or with some degree of aphasia when the left hemisphere is involved in right-handed persons. The proximal joints of the limbs and trunk show little or no loss in limited lesions of the parietal cortex. Putting it broadly, in the matter of spatial sense the middle two- or perhaps three-fifths of the posterior central convolution is the center

for the hand, the little finger being represented highest and the thumb lowest, but there is no evidence that the middle finger is represented midway between the two, and of the elbow and shoulder one can only say that they are likely to show sensory loss in gross lesions. Comparisons of more-or-lessness and judgments of textures are most involved in lesions lying along the lateral sulcus, but there is no evidence of definite localization of the body members in this region; in fact, the hand only lends itself readily to tests of this nature.

It will be seen that as in motor so in sensory lesions of the cortex the type of distribution is monoplegic. It remains to be said that sensory loss due to cortical lesions can be tested only after the period of shock has passed; and that no stationary cortical lesion, after the period of shock has passed, causes total loss of the primary forms of sensation in any part of the body. Patients suffering from limited lesions of the sensory areas of the cortex tire very quickly; their answers become uncertain and subject to wide variations while they are examined on the affected members, though they may be quite certain, prompt, and reliable in their replies to similar questions about the unaffected side. (Read the description of Figure 221.)

4. *Motor loss in lesions of the sensory cortex.*

(*a*) *Ataxia.* In proportion as a limb has lost the power of postural recognition the patient shows ataxia in that limb. This ataxia, like locomotor ataxia, is increased by closing the eyes, and is less marked if the patient can guide his movements by sight.

(*b*) *Hypotonia.* Wherever there is marked cortical loss of postural sense the limb involved shows a hypotonic condition of the muscles. If, however, there be simultaneous involvement of the anterior central gyrus, the hypotonia may be masked by the rigidity which accompanies the upper motor neuron paralysis, so that hypotonia is readily recognized only in purely sensory lesions.

SENSORY LOSS IN SUBCORTICAL LESIONS IN THE PARIETAL REGION

Simultaneous involvemement of more types of sensation and more overlapping of the loss in the hand and foot, and in the hand and face are characteristic of subcortical as contrasted with cortical lesions. In the left hemisphere concomitant aphasias are likely to accompany sensory loss in the hand and face when this is due to subcortical lesions.

A rather deep lesion of the left supramarginal gyrus may cause aphasia with apraxia.

SENSORY LOSS IN CAPSULAR LESIONS

The blending of pyramidal and thalamocortical fibers in the knee and posterior limb of the internal capsule makes it impossible that a capsular hemiplegia should be without sensory loss. This is, however, masked by two things, viz.—the thalamic recognition of the primary sensations of touch, pain, heat and cold, and the difficulty of testing postural sense in a hemiplegic. Nevertheless, careful examination will always show some sensory loss in capsular hemiplegics; postural sense at all joints suffers most (see Figs. 218, 219 and 222). Where the retrolenticular part of the capsule is involved there is homonymous hemianopsia.

MOTOR PARALYSES

PARALYSES DUE TO MUSCULAR LESIONS

(Classification, Part III, Supplement, page 293)

Paralyses not associated with nervous lesions but clearly myopathies are mentioned here only for the sake of completeness. In all the myopathies the diagnosis depends largely upon distinguishing between progressive weakness and emaciation due to disease invading the muscles themselves and weakness and atrophy secondary to a widespread involvement of the lower motor neuron cells.

The presence of fibrillar twitchings, together with electric reactions of degeneration in disease of the lower motor neuron cells, the absence of these in the myopathies, and the presence of myopathic electrical reactions, together with the history of the case and the distribution of the paralyses, are the main guides in diagnosis.

LOWER MOTOR NEURON PARALYSES

(Classification, Part III, Supplement, page 293; also Figs. 99 and 100)

Lower motor neuron paralysis may be defined as paralysis due to interruption of lower motor neurons anywhere from their cells in the anterior gray column of the spinal cord or brain stem to their terminal end organs in striped muscles. It also includes paralysis from the interruption of preganglionic neurons of the autonomic nervous system and parasympathetic efferent neurons. For a summary of the anatomy and physiology of the lower motor neuron see Part II, pages 125 to 127. This discussion will deal only with affections of the nerve cells and their axons, that is, anterior horn cells, motor nerve roots, and peripheral motor nerves.

A general survey of the forms of lower motor neuron paralysis in the

classification (Part III, Supplement, p. 293) shows that, in all, the essential feature is that the muscles involved are cut off from all nervous influences which normally flow constantly to voluntary muscles through this, their only connection with the central nervous system (Fig. 100). In proportion to the completeness and extent of the lesion the result is functional death and ultimately histological disintegration of the muscle or muscles. Tone, firmness to the touch, contractile power, electrical reactions, histological characteristics all ultimately disappear. If the nerve lesion be complete the muscle at once is powerless. Within a week it becomes flabby and begins to waste. Characteristic electrical reactions of degeneration (R.D.) appear in the first week and are established by the end of the second week; soon the muscle fibers lose their striation, the fibers shrink, they become hyaline and sometimes fatty, the fibrous elements tend to proliferate, and the entire muscle may disappear or be largely replaced by fat or fibrous tissue. For a very considerable time, however, after the nerve lesion, the muscle is capable of regaining its integrity if the nerve recover, or, in the case of a wound, be successfully sutured. Meantime the whole neuron undergoes rapid fatty degeneration if the original disease be in the nerve cell; or if the nerve trunk be injured the distal portion undergoes rapid fatty and then fibrous degeneration; the proximal part degenerates more slowly; the nerve cells show chromatolysis and ultimately disappear.

As the classification shows, the peripheral nerve is subject to various forms of inflammation, tumors, or traumatism; the primary nerve trunk, to pressure from affections of the vertebræ and from traumatism, the roots, to syphilitic radiculitis, to local meningeal inflammations, and to tumors (Figs. 184, 185 and 191). The nerve cells in the anterior gray columns may be affected by poliomyelitis, acute (Fig. 153) or chronic, by simple progressive wasting perhaps from congenital lack of resistance to ordinary wear and tear, as in progressive spinal muscular atrophy (Fig. 160); the cells may also suffer destruction by tumor or hemorrhage.

The distribution of the paralysis varies according to the site of the lesion. As most muscles are supplied by two to three segments of the spinal cord, at least two neighboring segments or two spinal nerves must be involved before a definite group of muscles is completely paralyzed. Thus, reference to Figure 95 and to the table in the supplement shows that the fourth, fifth and sixth cervical segments or nerves must all be involved before the deltoid is completely paralyzed, and the eighth cervical and first thoracic segments or nerves before the small muscles of the hand

lose their power; the same paralysis may result from stab wound of the upper trunk of the brachial plexus (fifth and sixth cervical) or the lower trunk (eighth cervical and first thoracic) of the brachial plexus. Where the lower motor neuron cells are involved in poliomyelitis, the paralysis will affect the muscles supplied by the cord segments; the same holds good for the individual spinal nerves (see Fig. 95); but when a peripheral nerve is injured, the muscles supplied by branches given off below the nerve injury will be those affected. For example, when the radial (O. T. musculospiral) in the spiral groove is involved in a fracture of the middle third of the humerus, it will be the muscles supplied by branches of the radial below the injury that will suffer, namely, the brachioradialis and the extensor group of the forearm; the brachialis will escape because of its double nerve supply. In poliomyelitis there is as a rule no anæsthesia, though there might be a segmental loss of pain, heat and cold owing to involvement of the second sensory neurons for pain, heat and cold, as the cells, synapses, and decussating neurons of these commence in and pass through the spinal gray matter (Fig. 224). On the other hand, injury to a spinal nerve involves sensory as well as motor loss. When the destruction of the anterior gray column cells is due to central gliomatosis as in syringomyelia (Figs. 164, 187 and 188) there is a characteristic type of anæsthesia accompanying the atrophic paralysis. For the sensory considerations see Classification. A few typical examples will serve to illustrate the general statement.

Poliomyelitis. A case of acute anterior poliomyelitis is illustrated in Figure 78; the oblique shading in the sections of the sacral region of the cord and of the cervical enlargement shows the areas of greatest destruction by the disease. In these sections the degeneration of the anterior nerve roots is well demonstrated by the stain, as the patient died three weeks after the onset of the disease, when fatty changes are marked. Figure 153 shows the late shrinkage of the affected gray column in an old case of this disease. Figure 155 is from an instantaneous photograph of a severe case of acute anterior poliomyelitis affecting chiefly the lumbar enlargement. The photograph, *taken while the child was struggling*, shows well how the legs hang flaccid, helpless and flail-like. The toes are pointed by the weight of the feet. The wasting has not yet developed, or, as is often the case in children, is masked by the subcutaneous fat. Contrast with this the false rigidity in the case illustrated by Figure 157. Here the iliopsoas muscles are paralyzed (supplied by L. 1 and 2), and the extensors of the thighs (glutei, hamstrings; L. 4 and 5, S. 1) have

escaped and are unopposed. On the other hand, in the child shown in Figure 159 the glutei and hamstrings are paralyzed by poliomyelitis in the fourth and fifth lumbar and first sacral segments, and the pelvis cannot be extended on the thighs. In this child the upper lumbar segments have escaped and the unopposed iliopsoas has bent the pelvis and lumbar spine on the thighs. As a consequence the patient can only attain the erect posture by an enormous compensatory backward curve of the spine.

In all these cases the disease is bilateral, but it is not always so. Figure 158 shows late bulging of the flaccid atrophied muscles in the right upper segment of the abdominal wall due to poliomyelitis in the right half of the seventh, eighth, and ninth thoracic segments of the cord. Figure 154 illustrates the late atrophy and failure in growth, and collapsed right side of the thorax due to an early poliomyelitis of the right upper segments in the cervical enlargement and right upper thoracic segments of the cord. Figure 156 is from a photograph of a patient with poliomyelitis of the nucleus of the right facial nerve. The boy is closing his eyes and screwing up his face vigorously; while the right eyelid closes it does not close so tightly as on the left side. The upper part of the right facial nucleus has partly escaped, but the lower part is profoundly affected, so that the right side of the lower part of the face is quite smooth in spite of most vigorous efforts, and the unopposed muscles of the left side are pulling the mouth and nostrils over to the left. This facial paralysis, coupled with a flaccid paralysis of the right shoulder, favors a diagnosis of polio-myelo-encephalitis as against other possible forms of facial palsy. If the facial nerve roots were affected the upper part of the face on the affected side could not contract at all.

Acute poliomyelitis is not the only disease that may attack lower motor neuron cells. Figure 186 shows a section through the sixth cervical segment of the cord of a case of *amyotrophic lateral sclerosis*. Only two or three faintly stained cells are found in each anterior gray column, in marked contrast with the numerous cells in the normal cord placed beside it for comparison. Figure 160 is from a section of the cord of a similar case, with Figure 161 from a normal cord for comparison. Figure 162 is from a photograph of such a patient, showing paralysis and extreme wasting of the forearms, hands and chest muscles. There are several distinct types of this disease.

In certain chronic affections of the lower motor neuron cells there is concomitant sclerosis of the lateral cerebrospinal tracts, so that both types of paralysis coexist. Under these circumstances if the disease be concen-

trated in the cervical region and the anterior gray columns escape in the lumbosacral enlargement, such a lesion (Fig. 186) may cause flaccid paralysis of the arms owing to the disappearance of the lower motor neuron cells in the cervical enlargement with spastic upper motor neuron paralysis of the legs due to the concomitant lateral sclerosis.

In the lesions considered so far no sensory cells or axons have been involved, and pain and anæsthesia have been absent. When, however, the lower motor neuron cells are destroyed by disease that also involves sensory neurons, the case is different. Figure 163 is from a case which is superficially not unlike Figure 162, but while the patient in 162 had no anæsthesia, Figure 163 has loss of pain, heat and cold down the medial sides of both upper limbs (Fig. 215). Paralysis and wasting of both forearms is well shown in Figure 216. Here the disease is a *gliomatosis of the gray matter with cavity formation* (Fig. 164) destroying both the lower motor neuron cells and the cells and commencing axons relaying pain, heat and cold for the seventh and eighth cervical and first and second thoracic segments of the spinal cord (compare Fig. 164 with Fig. 69). Figure 163 also shows atrophy of the muscles of the upper chest and back.

Anterior nerve roots (Fig. 99) and motor roots of cranial nerves. A radiculitis affecting the anterior nerve roots alone without involvement of the posterior nerve roots is unusual; while a syphilitic radiculitis of the posterior nerve roots alone is not uncommon, probably because of the exposed position of the posterior root ganglia. The roots of cranial motor nerves on the other hand are very vulnerable, owing to their long course through the brain stem, through the subarachnoid space at the base of the brain, and through the dura; in all these situations they are very much exposed to involvement in disease of neighboring parts.

Figure 180 shows how a paralysis of the oculomotor nerve on the side of the lesion may be accompanied by a hemiplegia of the opposite side of the body, and Figure 181 shows how the oculomotor paralysis may be accompanied by a hemianæsthesia of the opposite side of the body. In each case a single lesion causes very diverse symptoms. Figure 183 and Figures 174 to 179 show similar combinations. Again a tumor of the temporal pole of the right hemisphere may involve secondarily several or all of the nerves in the wall of the cavernous sinus by spreading to the dura at the base. Owing to its long intracranial course the sixth cranial nerve is frequently involved in tumors at the base of the brain.

The nerve roots that form the cauda equina are often involved by trauma or tumor. Here in case of tumor the pains are often severe, but

in crush the motor symptoms predominate. Figure 207 illustrates such a case.

Spinal nerves. Where spinal nerves are involved near their exit from the intervertebral foramina before they have united with others to form plexuses (Fig. 99; B. 2 of B. 2′), the symptoms are segmental in distribution and sensory loss accompanies the paralysis. Where one nerve only is involved close to its origin pain may be marked in incomplete lesions, but neither motor nor sensory loss may be noticeable; but where two adjacent nerves are affected both forms of loss may be marked. In Figures 210, a, b, c, and d, is illustrated a case of bilateral paralysis of the fifth and sixth cervical nerves (the upper trunk of the brachial plexus) due to bilateral cervical ribs. In this affection it is more often the eighth cervical and first thoracic nerves which suffer. Figure 210, d, is a rough sketch of the radiogram showing the accessory ribs. Figures 210, b and c, show the areas of pain and diminished sensory conduction; on comparison with Figure 96, a, b and c, these are seen to correspond fairly closely with the sensory distribution of the fifth and sixth root areas. The muscles paralyzed are the biceps, deltoid, brachialis, brachioradialis and supra and infraspinati; reference to Figure 95 and to the table showing muscular supply shows that these muscles are supplied by the fifth and sixth cervical segments. Thus the trunks formed by the fifth and sixth cervical nerves were evidently suffering from the pressure of the abnormal ribs. Rapid improvement followed operation on the right side so that there cannot have been any actual disorganization of the nerve trunks. It is not so stated in the case report, but doubtless an equally successful operation was performed on the left side.

Figure 166 shows a baby suffering from traumatic palsy of the extensors of the wrist and fingers, the long flexors of the fingers and intrinsic muscles of the hands. The anæsthetic areas are not mentioned. The injury was due to an attempt in forced delivery to draw the arm down by a finger hooked in the axilla. If the triceps escaped the injury was probably due to traction on the seventh and eighth certical and first thoracic nerves as they pass through the intervertebral foramina. If the triceps was involved the radial, median and ulnar nerves were probably injured in the outer wall of the axilla (see table).

Figure 151 illustrates paralysis of the radial (musculospiral) nerve where it crosses the medial border of the humerus; pressure was made on the nerve against the bone while the patient slept with the arms hanging over the back of a chair. The extensors of the wrist and fingers are

paralyzed. The triceps escaped as it is supplied by the radial nerve before it enters the groove. The sensory symptoms in such a case are limited to some numbness and tingling and diminished conduction over the back of the outside of the forearm and back of the hand. Similar pressure injuries occur in crutch palsy where the axillary (circumflex) nerve is usually affected; and when during the administration of an anæsthetic the arm is allowed to hang in a faulty position, injuring the radial or the ulnar nerve as it crosses the medial border of the humerus.

Figure 86 illustrates the anæsthesia and paralysis produced by traumatism of the ulnar nerve, and Figure 152 the late trophic changes which occur many years after division of the median and ulnar nerves near the wrist. Here all the intrinsic muscles of the hand are wasted, the second and third phalanges are flexed because of the loss of the interossei and lumbricals, the thumb is abducted by the unopposed long abductor; its adductors are lost. The trophic changes show most in the nails.

Peripheral neuritis. Peripheral neuritis, a type of paralysis that follows the distribution of no cord segment, nor any single nerve or combination of nerves, is illustrated in Figures 165 and 209. The distribution of peripheral neuritis appears to be frequently but not invariably determined by the distance from nerve or vascular centers; more or less symmetrically the extremities of all the limbs are affected. Sensory symptoms are prominent except in lead palsy, where, curiously, the sensory neurons are frequently spared. Figure 165 shows the typical wrist and foot drop of alcoholic multiple neuritis, and Figure 209 shows the sensory loss for all forms of sensation in a case of post-influenzal polyneuritis. Here the sensory symptoms were accompanied by flaccid paralysis with atrophy of all four limbs.

UPPER MOTOR NEURON PARALYSIS

Upper motor neuron paralysis is paralysis due to destruction of the large pyramidal cells in the motor area of the cerebral cortex (anterior central convolution with its extension on the medial surface of the hemisphere), or paralysis caused by interruption of the axons of these cells.

The anatomy and physiology of the upper motor neurons has been treated in Part II. The upper motor neuron appears to have a double function; it transmits voluntary impulses to groups of lower motor neurons so as to produce definite voluntary movements, and it controls spinal reflexes. This conclusion seems warranted because the prominent symptoms of a pure pyramidal lesion are loss of voluntary motion and increase

of tendon reflexes. It is probably more correct to assume that the upper motor neurons attain their results by using the whole infrapyramidal motor mechanism (lower reflex arc, spinal association system, cerebellar system, and basal ganglia system). Man, more than all other animals, suffers from the effects of a lesion of the pyramidal system, because in man it is most highly developed.

For convenience the term pyramidal neuron will be used as synonymous with upper motor neuron, and pyramidal tract as meaning the tract formed by the upper motor axons. In a pure pyramidal lesion the lower motor neuron is still under the influence of the other efferent systems and the spinal reflex mechanism (see Fig. 100). With the exception of limited lesions of the motor cortex cerebri, some subcortical affections, the rare and even problematical disease, primary lateral sclerosis, and perhaps distinctly limited lesions in the basis mesencephali (Fig. 180, a) there are no known lesions limited to the pyramidal system. The basilar pontine lesion in Figure 182, which at first seems limited to the pyramidal tract in its passage through the pars basilaris pontis would almost certainly involve cortico-ponto-cerebellar fibers. Such lesions will be discussed later.

THE DISTRIBUTION OF THE PARALYSIS

(*a*) *Group Control.* The pyramidal system controls groups of muscles, not individual muscles, through combinations of lower motor neurons, not through individual lower motor neuron cells, or even through individual cord segments; the influence is in some cases even more an inhibition of a muscle group than activation of its antagonists. Thus the action of the will on the sphincter ani or on the voluntary sphincter of the bladder is mainly inhibitory; tonic control of bladder and bowel is for the most part a cord reflex. Since the pyramidal control is not individualized, it follows that no group of muscles is completely paralyzed by an upper motor neuron lesion. They are only incapable of being activated directly for voluntary movements. This is in sharp contrast with lower motor neuron paralysis in which the affected muscle is cut off from all nervous influences, voluntary, automatic, and reflex, and rapidly wastes and degenerates.

In pyramidal lesions the muscles are still subject to many reflexes, are even too much subject to reflexes, and may often be used in synchronism with muscles of the well side. In a patient paralyzed in the left arm, the paralyzed forearm and hand may move involuntarily when a vigorous effort is made with the right unparalyzed arm.

(*b*) *Crossed Supply.* The chief pyramidal supply is always crossed; there are said to be exceedingly rare exceptions to this rule which are still obscure and need not concern us. Muscles of the two sides of the body which habitually act together have a weak homolateral pyramidal supply as well as the usual crossed connection. This applies to the upper face muscles, the muscles of the tongue, the muscles of mastication, those of the pharynx and larynx, the intercostal and abdominal muscles, the perineal muscles, and perhaps some muscles of the shoulders and hips. Careful examination will always show that if their crossed pyramidal supply is destroyed these muscles are weaker than the corresponding ones of the unaffected side. Thus a patient with upper motor neuron paralysis of the left side of the body can close his left eye with the right one, but cannot close his left eye separately nor can he close it so tightly as the other. He apparently protrudes his tongue straight, but if told to protrude the tongue vigorously, it is protruded toward the paralyzed (left) side because of weakness of the pyramidal supply to the left genioglossus. Similarly the left side of the chest moves synchronously with the right side, but on vigorous chest expansion will be found to expand less fully than the healthy side. In this respect there is a complete contrast with lower motor neuron paralysis which is always homolateral and is always complete for the nerve and muscle affected, except of course in limited lesions where the paralysis is in proportion to the extent of the lesion, as for example in progressive muscular atrophy from gradual loss of lower motor neuron cells. Here healthy fibers may be side by side with fibers in various stages of degeneration.

(*c*) *Skilled Movements Suffer Most.* The voluntary motor cortex of the cerebrum is most associated with skilled movements; many stock movements have an automatic infracortical mechanism in the cerebellum-corpus-striatum complex. Therefore in limited pyramidal lesions of the cerebrum, it is the forearms and hands and the feet which suffer most severely; the roots of the limbs frequently show considerable possibility of movement, especially synkinetic movements, that is, movements associated with forcefully willed movements of the well side. In lower animals the infracortical mechanism may very largely compensate for pyramidal loss—very much more so than it does in man.

(*d*) *Head and Eyes Escape.* In an acute pyramidal lesion in the cortex or internal capsule after the initial shock has passed off, the eyes escape without paralysis, and move synchronously for all conjugate movements. This is not due to a homolateral as well as a crossed supply, but is due

to automatic control by lower centers, probably the superior colliculi. During the early shock of an acute cortical or capsular lesion the head and eyes may suffer paralytic deviation *toward* the side of the *lesion* and *from* the *paralytic side* of the body. This is because one function of the posterior end of the middle frontal convolution is to turn the head and eyes to the opposite side, and in the early stages of an acute cortical or capsular lesion this function of the well half of the cerebrum is unopposed.

(*e*) *Relation to the Pyramidal Decussation.* Where the pyramidal lesion is above the pyramidal decussation, the paralysis affects the side of the body opposite the lesion; the lower face is included if the lesion is above the lower end of the pons. Where the pyramidal lesion is below the decussation the paralysis is on the same side as the lesion.

(*f*) *Bulbar Paralysis.* Owing to the limited area and the intimate relation of the blood supply of the two sides a one-sided vascular lesion of the pyramid in the medulla oblongata is very rare; such a unilateral lesion is depicted in Figure 174. Usually vascular lesions in this situation are more or less bilateral and a paraplegia results with added lower motor neuron paralysis of both sides of the tongue; most such lesions are immediately or rapidly fatal due to the involvement of vital centers. True bulbar paralysis is usually a bulbar extension of an amyotrophic lateral sclerosis or a polioencephalitis.

(*g*) *Cord Lesions.* Most cord lesions are bilateral and produce a paraplegia involving the legs alone or the legs and more or less of the trunk if they are below the cervical enlargement, or involving the arms, trunk and legs if at the level of the cervical enlargement. A lesion at or above the third or fourth cervical segment is immediately fatal from paralysis of the diaphragm.

(*h*) *Mixed Nature of Most Cord Lesions.* There are few cord lesions which are pure pyramidal lesions; the rubrospinal, the posterior spinothalamic, and the spinocerebellar tracts, and the posterior columns—any or all of these are usually also involved, and the vestibulospinal tracts, less frequently. Unless a cord lesion is complete, or at least unless the sensory tracts as well as the voluntary motor tracts are involved the bladder and bowels do not give serious trouble, except of course when the third and fourth sacral segments are injured; here again there is a mixed lesion.

(*i*) *Brown-Sequard Syndrome.* Destruction of one half of the cord by concussion, stab wound, or the pressure of a tumor causes a hemiparaplegia with definite sensory and lower motor neuron symptoms which will receive special consideration under the caption "Brown-Sequard syn-

drome." In one-sided lesions of the cord the bowels and bladder usually escape.

Rigidity or spasticity in upper motor neuron lesions. The whole subject of rigidity in upper motor neuron lesions has been carefully discussed under "general considerations," Part II. Here one or two points only will be recapitulated.

(*a*) *Flaccid Period.* Every acute pyramidal lesion of any extent whether in the brain or cord is followed by an initial period of flaccidity of the paralyzed parts lasting for a varying time. A capsular hemorrhage may be followed by flaccid paralysis of the affected side of the body; three weeks to three months may elapse before rigidity appears. In acute cord affections, softening, localized infective myelitis, or traumatism, it is seldom less than three weeks before cord reflexes of the paraplegic limbs appear; the patient may die before this occurs.

(*b*) *Pure Pyramidal Lesions.* Pure cortical lesions or pure pyramidal lesions just beneath the cortex, in which only the pyramidal control of cord reflexes is removed, are as a rule accompanied by very little rigidity and minimal contractures.

(*c*) *Capsular Lesions.* A capsular lesion, involving both the pyramidal and lentiform control of cord reflexes, results in the most typical form of spastic hemiplegia.

(*d*) *Lateral Sclerosis.* The lateral scleroses in the cord (Figs. 170 and 206) almost necessarily involve the rubrospinal tracts as well as the lateral cerebrospinal tracts; the vestibulospinal tracts escape. As the pallidospinal connections are through the rubrospinal tracts and perhaps partly by way of fibers from the corpus subthalamicus and substantia nigra mixed with the pyramidal fibers, it follows that a lateral sclerosis removes both pyramidal and pallidal control over cord reflexes and over the vestibulocerebellar tonic impulses which travel by the vestibulospinal tracts and therefore the paraplegia of lateral sclerosis is markedly rigid in type.

(*e*) *Chronic Lesions.* Chronic, slowly progressive lesions, such as some tumors, are accompanied by no period of shock; they are spastic in type from the beginning in proportion to their distribution.

Reflexes. Reflexes have been fully discussed under "general considerations," but the chief points bearing on their relation to pyramidal lesions may be here recalled.

(*a*) During the period of *flaccidity* all reflexes are lost. The earliest to appear when the leg centers or tracts are involved is the Babinski phenomenon.

(*b*) *Skin reflexes* are usually lost for the part involved in pyramidal lesions.

(*c*) The *Babinski phenomenon* (an extensor response of the great toe when the lateral margin of the sole of the foot is stroked) is pathognomonic of a pryamidal lesion in cases in which the legs are affected. In infants before walking age it may be normally present owing to the undeveloped condition of the pyramidal tracts. It is the first reflex to appear as "shock" wears off and may appear within a few hours in apoplectic cases. It is absent if there be any concomitant lesion affecting the reflex arc (as in tabes), and it is never found in hysterical paralysis. In complete cord lesions the plantar response is either absent or flexor in character during the period of shock. One of the earliest signs that shock is wearing off is the appearance of an extensor plantar response. This is usually accompanied by automatic emptying of the bladder.

(*d*) The *foot adduction reflex* of Marie and Meige is said to be characteristic of cortical lesions. If the median side of the foot be scratched the foot is adducted and medially rotated.

(*e*) *A thumb reflex* in hemiplegia and brachial monoplegia is allied to the Babinski reflex, and is probably as delicate and as distinctive of pyramidal involvement. It is physiologically present in children up to two and a half years; after this it is pathological. If the skin along a narrow strip of the hypothenar eminence be stroked with a blunt point the thumb is adducted (*Revue Neurologique,* May, 1923, page 506).

(*f*) *Tendon reflexes* are always exaggerated in pyramidal lesions except in some cases of concomitant disease as in tabes dorsalis, pellagra, and diabetes.

Concomitant phenomena. As so few lesions affect the pyramidal system alone, symptoms of lesions of neighboring centers or tracts are always to be expected. These may be of the nature of sensory loss, lower motor neuron paralyses, lenticular or cerebellar symptoms. Such concomitant symptoms may be of the utmost value in fixing the site of the lesion. Here the following points are to be remembered.

(*a*) Athetoid and choreiform movements (Figs. 200 and 201) and tremors are only possible when pyramidal disease affecting the parts involved is exceedingly slight. They are almost certainly due to concomitant lesions of the neostriatum, or perhaps of the thalamus.

(*b*) Cerebellar symptoms are almost entirely masked in paralyzed limbs, but in pyramidal lesions in the mesencephalon or pons cerebellar

symptoms may sometimes be expected to affect the limbs on the unparalyzed side. (See pontine lesions.)

SOME TYPICAL PYRAMIDAL LESIONS AND THEIR SPECIAL FEATURES

Cortical lesions. From an examination of Figures 90 and 91, where the whole motor area of the cerebral cortex is drawn approximately life size, it will be evident that a rather constant feature of an upper motor neuron paralysis which is due to a cortical lesion must be its limitation to one member. Thus a lesion of the lower end of the left anterior central convolution would cause a right facial monoplegia; a lesion of the middle third would cause a right brachial monoplegia; and lesion of the upper third a paralysis of the right leg; or an intermediate lesion might produce a facio-brachial combination or a brachio crural combination; but a single lesion could never cause a facio-crural combination, with the arm unaffected. *A cortical lesion then is always a simple monoplegia or a combined monoplegia of the facio-brachial or brachio-crural type.*

Jacksonian epilepsy. In all the length of the pyramidal tract the cortical cells only are subject to irritative symptoms producing convulsive seizures. A cortical convulsion due to irritation by a localized cortical lesion, such as a cortical or meningeal or cranial tumor or spicule of bone caused by fracture, is called Jacksonian epilepsy. Its essential characteristic is that it always starts with the same member and spreads to others in a definite order, as do the circles from a stone thrown into a pool. For example, referring again to Figures 90 and 91, a fit starting with flexion of the fingers of the right hand would be followed by flexion of the wrist and elbow simultaneously with turning of the head and eyes to the right, then shoulder and hip movements would follow simultaneously with movements of the jaws and lips. Finally the convulsions spread to the opposite side of the body. Consciousness is seldom or never lost in Jacksonian epileptic seizures. Spreading to the posterior central convolution it may cause numbness and tingling of the convulsed parts. A fit starting in the toes of the right foot would spread to the hip and shoulder before affecting the head, eyes, and jaws. By careful observation of the order of invasion the site of the tumor may be localized near the center for the muscles in which the fits *invariably start*. Further, a lesion causing paralysis of the center where it is actually located may cause Jacksonian fits referred to the parts supplied by neighboring motor and sensory areas. A tumor of the middle of the left posterior central con-

volution may commence with a numbness or tingling of the fingers, followed by flexion of the fingers, and wrist, spreading further centrifugally as before.

Sensory aura. By a study of Figure 90 in relation to Jacksonian epilepsy it will be evident from the location of the auditory center that a Jacksonian fit due to tumor in this neighborhood may start with a subjective sensation of a sound and spread to the face and arm, causing tingling and convulsions of the right side of the face and right arm. A tumor in the posterior end of the left inferior frontal gyrus which in right-handed people is the center for motor speech memories) may associate more or less motor aphasia with convulsive attacks beginning in the tongue, lips, and face and spreading to the right arm and leg in this order.

LESIONS DUE TO BLOCKING OF CORTICAL VESSELS

Middle cerebral artery. Passing to the consideration of vascular lesions affecting the motor cortex, study of Figures 146, 142 and 143 shows that an embolism in the trunk of the left *middle cerebral artery* after it has given off its central branches may cause widespread cortical paralysis involving the right half of the head and the right arm, but the right leg, whose cortical center is supplied by the anterior cerebral artery (Fig. 167), will escape. In right-handed people the centers for auditory word memories are in the left superior temporal convolution and the centers for motor word memories, or the educated mechanism of spoken speech are in the left inferior frontal gyrus (Fig. 142); therefore, in right-handed people an embolus of the lateral trunk of the middle cerebral artery may be associated with sensory and motor aphasia (loss of the understanding of spoken speech or loss of the power to speak), together with paralysis of the right side of the face and the right arm. A single lesion can never produce paralysis of the right leg combined with aphasia, either auditory or motor.

Anterior cerebral artery. A thrombosis or embolism of the left *anterior cerebral artery* may produce a monoplegia of the right leg; with this there may be combined corpus callosum symptoms, apraxia of the left hand without paralysis, which will be considered later. Vascular lesions of the cortex are not usually so extensive; smaller branches of the vessels are more often affected and the resulting monoplegia involves the hand and forearm alone, or speech alone, or speech and the right side of the face together. In left-handed people the right hemisphere is usually educated for speech and the higher motor memories; in them it is a left

brachio-facial monoplegia that is associated with loss of speech (aphasia), but this is not an invariable rule. (Compare Figs. 142 and 144).

To summarize, a cortical paralysis is usually a monoplegia; it may be associated with Jacksonian fits, and a cortical facio-brachial monoplegia of the right side in a right-handed person may be associated with aphasia. If the onset is sudden it is probably of vascular origin, and if of slow progressive nature it is probably due to some form of tumor. It may also be associated with the cortical type of anæsthesia (impairment of sensory judgments without actual loss of sensation). Rigidity is not of marked degree.

SUBCORTICAL LESIONS

The distribution (Fig. 148) of the central and cortical branches of the cerebral arteries, of which the penetrating branches are all terminal, that is, do not anastomose with each other, leaves an ischæmic region in the subcortical white substance which easily becomes bloodless and undergoes softening and necrosis if the circulation in the supply vessels be obstructed by arteriosclerosis. Such an area of softening in the neighborhood of the anterior central convolution may destroy pyramidal fibers from the motor cortex on their way to the internal capsule; it will be a pure pyramidal lesion except for concomitant destruction of subcortical association fibers. This is important with reference to the relation of subcortical softening to aphasia.

Some idea of the importance of these association fibers is given by lesion 10, in Figure 105. The whole mechanism of speech depends on an intimate association between the auditory speech center in the superior temporal convolution, the motor speech center in the inferior frontal gyrus, and the visual word center in the angular gyrus (Fig. 142). Now a subcortical softening in the neighborhood of the lower third of the anterior central convolution in the left hemisphere would be very likely to destroy association fibers between the auditory and motor word centers (Fig. 105, Lesion 10), and probably also fibers between the visual and motor word centers. Hence it is a common experience to find a right facial or facio-brachial monoplegia associated with a permanent aphasia; this is the ordinary type of subcortical pyramidal lesion in this region. In right-handed people the aphasia is only found in affections of the left hemisphere. Tumors also may commence subcortically, and the ischæmic area is rather a common site of abscess of low grade infective origin. Occasionally a subcortical tumor may give rise to Jacksonian epilepsy.

The pure pyramidal type of paralysis, with little rigidity (unless the putamen or globus pallidus be also involved), having a rather wider distribution than occurs in cortical lesions, together with frequent association of aphasia in right-sided facio-brachial paralysis are the chief characteristics of subcortical lesions.

ILLUSTRATIVE CASES

In Figure 79, a and b, is shown the area involved in a case of extensive *cortical and subcortical* softening from occlusion of branches of the middle cerebral artery. Death occurred within a month, and the resulting pyramidal degenerations are shown by the Marchi technique.

In Figure 167 the shaded areas show the sites of the lesions in various types of *cortical monoplegias;* the result of each lesion is given in the description of the figure.

A *left facial monoplegia* is shown in Figure 193. This was due to embolism in a branch of the middle cerebral artery occurring in a young woman with valvular heart disease. The lower third of the right anterior central convolution is affected. The lower face is paralyzed, while the upper face is only paretic. The paresis of the left upper face is shown by the fact that the left palpebral opening is greater than the right from weakness of the orbicularis oculi. In the original description by Dejerine this case is called a hemiplegia. A facio-brachial monoplegia would have been expected.

Figure 198 shows a child with *infantile cerebral monoplegia* due to a cortical affection of the middle third of the right anterior central gyrus, probably from polioencephalitis. There is no history with the figure (from Jelliffe and White), but the leg seems paretic. There appears to be slight rigidity of the elbow, enough to adjust the relative strength of the flexors, extensors, and weight. The fingers are not rigid. There is no wasting of the arm, though it is less well developed than the right.

A case of *congenital diplegia* of both arms and of both legs is shown in Figure 199. The patient was delivered by forceps, which probably caused cortical hemorrhages, chiefly in the upper thirds of both anterior central gyri. The face has escaped and the arms were improving at the time of the photograph; the legs were most seriously affected.

Figure 137 is from a photograph of a man aged 34 with diplegia due to double subcortical lesions affecting the fibers from the upper two-thirds of both anterior central convolutions. The disease commenced at the age of three months following an attack of convulsions. It is probable that

the convulsions caused double subcortical hemorrhages which resulted in double porencephaly. The face is unaffected, but there is considerable mental impairment. There was no epilepsy nor athetoid movements. The limbs are atrophied from disuse and the contractures proceed from long over-action of the stronger muscles and uncorrected faulty attitudes; the thighs are in extreme adduction.

CAPSULAR PARALYSIS

A large percentage of all "paralytic strokes" are due to softening of the internal capsule or hemorrhage into it. The result is a typical spastic paralysis of the opposite side of the body; the eye muscles escape entirely after the first shock has passed off, the orbicularis oculi and other upper face muscles are only weakened, and muscles acting in constant association on both sides of the body are only weak on the paralyzed side.

A *softening of the internal capsule* causing a hemiplegic attack one year before death is shown in Figure 168, a, and Figure 168, b, shows the resulting degeneration of the pyramid at the level of the decussation.

Figure 148 shows part of the distribution of the *lenticulo-striate artery.* This is the vessel of the anterolateral group of central arteries that is most frequently affected either by thrombosis causing softening, or by rupture causing hemorrhage in capsular paralysis. In most cases the globus pallidus is involved as well as the posterior limb of the internal capsule, and the removal of pallidal control of the tonic mechanisms adds to the rigidity.

Other vessels of the anterolateral group of central branches of the middle cerebral artery supply the anterior part of the lenticular nucleus, the head of the caudate nucleus, and the genu and anterior limb of the internal capsule. The anterior chorioidal artery is largely responsible for the supply of the middle part of the posterior limb of the internal capsule; the part of the capsule behind the lenticular nucleus (retro-lenticular part), and the optic thalamus are supplied by the posterolateral central branches of the posterior cerebral arteries. (Consult a picture of the cerebral vessels and Fig. 87, a, b, e.)

In Figure 87, e, it may be noted that the pyramidal fibers (dotted) have a certain order from the knee backward, but the areas overlap so much that capsular lesions are always hemiplegic, never monoplegic; still, a limited lesion far back in the capsule may eventually clear up, leaving only a facial paresis with more marked paralysis of the arm and leg. (See Figs. 196 and 197 for pictures of such cases.) The nearer one passes to

the base of the brain the more concentrated are the descending fibers of the internal capsule. A lesion near the dorsal part of the posterior limb of the internal capsule cannot avoid injury to thalamocortical fibers, hence most cases of capsular hemiplegia are associated with sensory loss of the hemiplegic area. This may require careful search as it does not affect the primary sensations of heat, cold and pain which reach consciousness in the thalamus. It is sensory judgments that are lost, sense of tactile localization, comparative judgments of texture, and passive position.

COMA AND EARLY FLACCIDITY

Unless a capsular lesion be very limited it is associated with a period of unconsciousness of varying duration. This is usually so in capsular hemorrhage, but there may be no comatose period in thrombotic softening. During this time the paralyzed side may be identified by anæsthesia of the cornea, flapping of the lips and cheeks in respiration, and greater flail-like helplessness of the paralyzed limbs when they are lifted and allowed to fall. During this period the tendon reflexes are absent; the plantar response, at first absent, soon becomes extensor (positive Babinski) on the paralyzed side. Other skin reflexes are absent on the paralyzed side.

Rigidity appears slowly in from one to three months after the onset; eventually in most capsular disease the paralysis is of marked spastic type. If the left capsule be the one involved in a right-handed patient there may be considerable difficulty in pronouncing words (dysarthria), but the patient knows perfectly what he wants to say; there is no true aphasia in a pure capsular lesion.

ILLUSTRATIVE CASES

As has been already noted, Figure 168, a, illustrates a typical capsular softening from which the patient recovered, dying one year afterward of a capsular hemorrhage. The section, and that of the pyramidal decussation (Fig. 168, a) showing the degeneration, are stained by the iron-hæmatoxylin technique for myelin, so that the areas of softening and of axon degeneration do not take any stain.

Figures 196 and 197 show typical hemiplegic cases after they have recovered sufficiently to be able to walk. The arm is more or less rigid in adduction and medial rotation, the forearm slightly flexed, the thumb adducted, and the fingers flexed (Fig. 194). There is no wasting, in fact,

the limbs may feel firmer than those of the opposite side from the unrestrained reflex activity of the muscles. The position is the result of the balance of weight and the comparative strength of the opposing muscles under the influence of unrestrained tonic mechanisms, cerebellar, vestibular, and spinal. The lower limb is rigid in extension at the hip, knee, and ankle, since the extensors are the stronger muscles and the preponderant cerebellar influence is extensor. The leg, rigid in extension, is available for walking, as an artificial leg would be. Motion is possible at the hip and is largely a swinging of the pelvis by trunk muscles and those muscles of the root of the limb that have a homolateral pyramidal or infracortical automatic supply. Figure 194 shows the typical attitude of a hemiplegic forearm and hand. Extreme rigid flexion of the upper and lower limb is rare in hemiplegia and is found only in old bedridden cases. The explanation of such cases is not very clear. It is the attitude of complete rest as it is usual in sleeping, and the attitude of the fœtus in utero, and may be the result of the exhaustion of the overworked and unrestrained tonic mechanisms.

MESENCEPHALIC AND PONTINE PARALYSES

Typical mesencephalic and pontine forms of hemiplegia of the basilar variety are illustrated in Figures 180, 182 and 183. Here the basis mesencephali or basilar portions of the pons alone are involved and therefore no sensory tracts or sensory symptoms complicate the resulting paralysis.

Mesencephalon. Figures 180, a and b, are typical examples of limited mesencephalic vascular lesions. As the medial lemnisci escape, there is no sensory loss. So far as is known the rubrospinal tract is the chief descending tract carrying lentiform control over muscle tone, and as this tract escapes in both cases they should be *pure pyramidal lesions* with minimal rigidity and contractures. The involvement to some extent of the lower motor neurons of the oculomotor nerve, combining more or less homolateral ocular paralysis with crossed pyramidal symptoms, is characteristic of mesencephalic lesions of the pyramidal tract. The varying positions of the aberrant pyramidal fibers in the mesencephalon on their way to the motor nuclei of cranial nerves accounts for some variability in the extent to which the opposite side of the face, the tongue, palate, pharynx, and larynx, and sternomastoid may be involved in these lesions. These aberrant fibers are exceedingly variable. They may occupy the medial ventral strip of the basis pedunculi (Fig. 79, c) and a rounded

bundle is frequently lateral to the substantia nigra. This explains how the palate, larynx, and sternomastoid escape in 180, a, but are involved in 180, b.

Upper pons. In the pons (Fig. 182) the aberrant pryamidal fibers to cranial nerve nuclei lie in the medial corner of the medial lemniscus, hence in 182, a, the left external rectus and the left sternomastoid escape, but in 182, b, they are involved by the extension of the lesion dorsally. The effect of the isolated lesion of the medial longitudinal bundle in 182, a, will not be considered at this time.

Lower pons. Figure 183 illustrates pure basilar lesions near the lower end of the pons; in 183, a, the destruction involves the emerging sixth cranial nerve, and in 183, b, it involves both the sixth and seventh nerves. These are examples of the typical lower pontine syndrome. Here also is an excellent opportunity to contrast the facial paralysis in 182, a, a typical *upper motor neuron* facial paralysis, in which the orbicularis oculi and frontalis muscles escape (compare Fig. 193), with that in 182, b, a *lower motor neuron* paralysis of the whole face including inability to wrinkle the forehead or close the eyelid. Moreover, in Figure 182 the facial paralysis is on the same side as the hemiplegia, while in Figure 183 the facial paralysis is on the *same side* as the *lesion,* and the hemiplegia is on the opposite side of the body.

In pure basilar pontine lesions the rubrospinal tracts escape and the rigidity should be minimal. Further, no extensive lesion of the basilar portion of the pons is possible without interruption of fronto- and temporo-pontine synapses to the opposite cerebellar hemisphere, and ponto-cerebellar fibers to the cerebellar hemisphere of the same side. Most cerebellar symptoms (asthænia, asynergia, adiadokokinesis, tremor) are completely masked by a pyramidal lesion; but as the fibers of the brachium pontis start in cells of the opposite side and intercross, a one-sided lesion of the basilar portion necessarily involves neuron cells for the opposite brachium and fibers before their crossing, as well as fibers for the brachium of the same side after crossing. Therefore in any lesion of the basilar portion of the pons cerebellar symptoms should be searched for on the side of the lesion, which, of course, would be the unparalyzed side. However, there is the difficulty that a patient with a pontine lesion is seriously ill, and much intelligent coöperation is necessary to enable one to diagnose even gross cerebellar lesions. The mixed basilar and tegmental pontine lesions shown in Figure 178, a and b, will be considered with sensory lesions.

Pyramidal lesions in the medulla oblongata limited to one side are exceedingly rare. Figures 174 and 175 illustrate two cases. The distinguishing feature in these two cases is the bulbar form of crossed paralysis, that is, lower motor neuron paralysis of the homolateral hypoglossal nerve simultaneously with upper motor neuron paralysis of the opposite side of the body. In Case 175 the vago-accessory group is included with the hypoglossal. In both cases there are accompanying sensory lesions; in 174 a lesion of the medial lemniscus caused loss of muscle sense and tuning-fork sense on the same side as the hemiplegia; and in 175 a fifth nerve anæsthesia on the side of the lesion with a crossed anæsthesia similar to 174. The combined symptoms accurately fix the site of the lesion in each case.

Acute bilateral pyramidal lesions in the medulla oblongata are rapidly fatal; probably in most cases too rapidly to admit of correct diagnosis. Chronic lesions will be considered later with amyotrophic lateral sclerosis.

SPASTIC DIPLEGIA

Before passing to cord lesions it is necessary to say a word about *double hemiplegia,* that is bilateral upper motor neuron paralysis due to lesions in both hemispheres.

Spastic diplegia, when it occurs in adults, is due to bilateral lesions; the strokes are usually separated from each other by a variable interval of time. Two separate strokes in an adult call for no special comment *unless both involve pyramidal fibers to bulbar nuclei.* If a vascular lesion occurs in the inferior end of each anterior central convolution, or one in this region of the cortex and the other in the opposite internal capsule, or one in each internal capsule, or if there be any other combination of lesions that interrupts both pyramidal tracts above the medulla oblongata so as to damage the pyramidal supply to both hypoglossal, both facial, both glossopharyngeal-vago-accessory nuclei, then the patient's breathing, swallowing, articulation, and mastication are very seriously interfered with and his condition is very pitiful. The condition is known as pseudo-bulbar paralysis. True bulbar paralysis, which is a lower motor neuron paralysis of bulbar nuclei on both sides, is usually a bulbar form of amyotrophic lateral sclerosis and will be described later.

Diplegia in children. Bilateral disease of the upper motor neuron in its cerebral segment is rather common in children.

(1) *Congential Cases—Little's Disease* or double hemiplegia of spastic type showing itself soon after birth in prematurely born children, may

be due to failure of the pyramidal tracts to develop. The pyramidal supply of the legs usually suffers most severely and more permanently. There is hope of improvement up to puberty (Fig. 199).

(2) Diplegia affecting both legs in children dating from birth may be due to *cortical or subcortical hemorrhage* occuring during delivery in the leg areas of both anterior central convolutions. These hemorrhages are especially liable to occur in breech presentations.

(3) Certain cases of rather widespread spastic diplegia occurring in children commence with fever and convulsions and may result from a polioencephalitis concentrated on the motor cortex. Such cases often show lack of mental development.

(4) Cases occur in which congenital or infantile rigidity is the prominent feature, or in which athetoid movements are the chief symptom (Figs. 200 and 201). In these the difficulty in using the limbs is mainly of the nature of difficulty in controlling them, owing to the rigidity or to the constant athetoid movements. In such cases there is probably a congenital or infantile affection of the basal ganglia rather than of the pyramidal system. Evidence is accumulating which seems to indicate that where athetoid movements are most in evidence the neostriatum (caudate nucleus and putamen) is affected; while the palæostriatum (globus pallidus) has suffered in the rigid cases. This explanation is still far from being established.

UPPER MOTOR NEURON PARALYSIS DUE TO CORD LESIONS

In most cases of lesion of the upper motor neuron in the spinal cord *both sides* are almost equally involved and the result is a *paraplegia.* Where the lesion occurs below the cervical enlargement both legs are spastic and paralyzed, but the arms escape; more rarely the arms also are more or less involved. Whether or not there be accompanying sensory or lower motor neuron symptoms depends on the nature of the lesion. Transverse lesions involving sensory as well as motor tracts, such as trauma, softening, transverse myelitis, or pressure by tumors of all kinds, belong in a class by themselves and will be considered separately.

Primary lateral sclerosis. The purest type of lesion of the upper motor neuron in the spinal cord is a primary sclerosis limited to the lateral cerebrospinal tract. It is a rare disease, so rare that it is doubted if such a cord would stand modern staining methods without showing other changes than this and the involvement of other tracts. The condition of

the cord at autopsy is somewhat like that in Figure 170, which illustrates an almost pure lateral sclerosis probably of syphilitic origin and accompanied by a diffuse endarteritis. However, in Figure 170 the posterior spinocerebellar and rubrospinal tracts are also involved, and the disease extended up to the pyramidal decussation, whereas primary lateral sclerosis is usually confined to the lower part of the cord.

The symptoms are typically those of a slowly progressive lesion of the lateral cerebrospinal tracts, though the spasticity is so extreme that the writer wonders whether the rubrospinal tracts, which are the main efferent tracts from the lentiform nuclei, may not have become involved in the sclerosis and so partly account for the spasticity. The rubrospinal tracts are so closely blended with the anterior part of the lateral cerebrospinal tracts that they could hardly escape the sclerosis following on the wasting of the cerebrospinal neurons.

The description of such cases, typical of a slowly progressive lesion of the cerebrospinal tracts, is quoted from Starr: "The symptoms of spastic paralysis (paraplegia) are very gradually increasing stiffness and rigidity of the muscles of the legs attended by an increase in reflexes and a tendency to cramps and tremor. The disease may begin on one side, but soon becomes bilateral. The patient appreciates difficulty in all motions of the legs. He cannot step freely, he cannot go upstairs with comfort on account of the great stiffness of the joints and muscles (see Figs. 203, 204 and 206). It requires a great effort to produce slight movements, and is as difficult as active voluntary motion.

The gait is characteristic of the affection (Figs. 203 and 204). The feet are not lifted from the ground, the toes are dragged, the shoe wears out on its inner border and toe, the legs cannot be abducted freely, and the knees have a tendency to overlap (Figs. 202 and 206). The patient shuffles along the ground, his steps become short, there being trepidation due to the increase of reflex action causing a clonus of the foot at every step. Much fatigue is felt on walking and the muscles often ache. Little by little the stiffness increases, until the entire lower extremity appears to be moved as a mass without any motion of the ankle or knee joints, and all efforts such as crossing the legs, kneeling down, or kicking are very much hampered and finally become impossible. The muscles appear to be made of hard tense cords, and offer resistance to any passive movements. Percussion upon the muscle or upon its tendon is immediately attended by a quick response, or even by severe twitchings or by a marked

clonus. Such twitchings and spasms may occur spontaneously and the patients complain of cramps and of twitchings which often interfere with sleep.

Ankle clonus appears early; a clonus is often obtainable in the toes, and downward pressure upon the patella or upon the adductor tendons of the thigh may elicit a clonus. Sometimes a sudden spasm of the legs causes a straightening out of the limb and adduction of the thighs. The Babinski reflex appears early. The muscles of the hip are not as early or as seriously affected as those of the knee and ankle; consequently the patient can walk for several years after the disease has developed, and can move the thighs in bed, even when unable to walk. But as the disease goes on and the patient is finally confined to the chair or bed, contractures of the affected muscles occur, the knees are drawn up and overlap (Fig. 205), the heels are drawn tightly against the buttocks, and the greatest efforts of the examiner fail to produce an extension of the legs. In this condition when the muscles cannot be actively or passively moved they gradually atrophy from disuse until finally the legs are reduced to a skeleton appearance, the few muscles left being still contractured. During all this period there are no sensory symptoms excepting general muscular pains and there is no disturbance of the bladder or rectum. There are no trophic changes unless toward the close of life long-continued pressure or lack of care results in the appearance of bed sores. The electric contractility of the muscles remains normal."

Figure 170, a to e, are drawings showing lateral sclerosis in a patient who died at the age of 70 years after having symptoms similar to those described for 30 years. Ultimately he became bedridden, his bladder began to give trouble and he died with evidence of acute myelitis. In this particular case a marked hyaline thickening of the walls of the blood vessels suggested a possible syphilitic cause for the sclerosis. Above the pyramidal decussation the brain stem was normal.

Erb's syphilitic paraplegia presents a somewhat similar picture, but usually runs a shorter course. The weakened cord is liable after some months to become the site of an acute infective transverse myelitis which quickly leads to death.

Combined sclerosis (Fig. 171) is usually associated with progressive anæmia; it runs a subacute but fatal course. As shown in the specimen (Fig. 171), the posterior columns, lateral columns, and even the anterior columns are involved; posterior column symptoms (loss of tuning-fork and compass sense, loss of passive position and stereognosis) are com-

bined with progressive paraplegia, and involvement of the posterolateral columns produces hyperæsthesia and hypæsthesia. The patient from whom this cord was obtained was for months treated for rheumatism of his legs.

When *multiple sclerosis* (Fig. 172) attacks both cerebrospinal tracts in the cord, paraplegia may be the most prominent symptom (Fig. 205).

In *Friedreich's ataxia* (Fig. 189) paraplegic symptoms are blended with those of cerebellar origin, and also with posterior column symptoms.

SIMULTANEOUS AFFECTION OF BOTH THE UPPER AND LOWER MOTOR NEURONS

AMYOTROPHIC LATERAL SCLEROSIS

Figure 186 illustrates the lesions found in amyotrophic lateral sclerosis. Here go side by side a slowly progressive sclerosis of the lateral cerebrospinal tracts and a gradual disappearance of the lower motor neuron cells in the anterior gray columns of the cord. The cases vary greatly in the relative severity of the two conditions, so that in some the sclerosis is more advanced and consequently spasticity is more marked, while atrophy, fibrillar twitchings, and reactions of degeneration proceed more slowly. In other instances as in the case illustrated (Figs. 186, 160 and 162) the loss of the lower motor neuron cells is more marked, and the slowly progressive muscular wasting is the preponderating symptom. Pain is slight throughout the disease.

The common form of *bulbar paralysis* is an amyotrophic lateral sclerosis affecting the medulla oblongata, where sclerosis of the pyramids, causing paraparesis, is associated with the disappearance of the cells of the hypoglossal, facial, and accessory motor nuclei. The patients die of diaphragmatic paralysis (as did the case illustrated; Fig. 162) or of the effects of the bulbar palsy; or they may die of some acute infection to which they show enfeebled resistance.

TRANSVERSE LESIONS OF THE SPINAL CORD, COMPLETE AND INCOMPLETE

(Read pages 193 to 195)

The individual afferent and efferent tracts of the cord have been described with the physiological functions and the effects of lesions of each tract. Before discussing the lesions involving more than one tract, *i.e.,* partial and complete transverse lesions of the cord, it is necessary to refer briefly to the *reflex and autonomic centers* in the cord and the *sympathetic connections of the latter.*

Figure 94 shows the location of the autonomic centers and the segmental relations of referred pain, and Figure 95 the deep and superficial reflexes and the segmental supply of some of the more important muscles. The segmental supply of all the muscles of the upper and lower limbs is given in a table at the end of the book. The data given in the paragraphs to follow regarding the autonomic centers are of special clinical importance in the diagnosis of cord lesions; the discussion should be read with constant reference to Figure 94.

Pupillodilator or so-called ciliospinal center (Figs. 94, 95). In the eighth cervical and first and second thoracic segments of the cord are cells which send rami communicantes by way of the first and second thoracic nerves to the corresponding thoracic ganglia of the sympathetic. They ascend through the cervical sympathetic, are interrupted in the cells of the superior cervical ganglion and by means of a new relay pass by the nasociliary nerve to the dilatator iridis and superior palpebral muscle of Müller, and by the maxillary nerve to the orbitalis muscle.

Injury to this ciliospinal center or to the cervical sympathetic causes miosis (*i.e.,* contraction of the pupil) because of the unopposed action of the spincter iridis supplied by the third nerve. Also the pupil fails to dilate in the dark, or reflexly when the skin of the neck is pinched. Narrowing of the palpebral fissure follows injury to the ciliospinal center with slight drooping of the upper eyelid because of the paralysis of the involuntary fibers in the upper eyelid (superior palpebral muscle of Müller). There is also recession of the eyeball owing to paralysis of the orbitalis muscle. The center in the cord is controlled by a higher center in the medulla oblongata (see page 210), and interruption of the cord anywhere between the spinal center and its bulbar control may cause similar pupillary contraction and drooping of the eyelid.

Sweating and tear secretion. In the second and third thoracic segments of the cord are cells situated in the intermediolateral cell column which represent an intercalated center in the sympathetic system for the control of sweating of the head and neck and arms, and the tear secretions. Partial injury to the cord here may cause dryness of the skin of the regions mentioned and dryness of the conjunctiva. Centers for sweating of the arms are in the fourth to the ninth thoracic segments, for the trunk below the umbilicus in the ninth or tenth thoracic to the third lumbar, and for the lower extremity from the eleventh or twelfth thoracic to the third lumbar. These centers are connected with a bulbar control by an

uncrossed tract, and partial injuries to the cervical cord higher up in the neck may cause similar symptoms (Gordon Holmes).

Cardiac center. Through the second and third thoracic nerves the upper segments of the thoracic cord also supply *white rami communicantes* which pass by way of the cardiac branches of the sympathetic and serve to accelerate and augment the heart beats. In soldiers wounded in the late war Gordon Holmes found that partial injury to the lower cervical and upper two thoracic segments of the cord occasionally caused rapid pulse (100-120-140 per minute).

Abdominal viscera. From the fourth thoracic segment downward to the second lumbar the white rami communicantes go to sympathetic ganglia controlling the *abdominal viscera.** Partial injury to the cord (by bullet or shrapnel for instance—Gordon Holmes) in the sixth, seventh or eighth thoracic segments may be accompanied by uncontrollable vomiting and girdle pains like the gastric crises in tabes dorsalis. Similar injuries between the fifth and eighth thoracic segments were sometimes accompanied by polyuria, probably due to paralysis of the vaso-constrictor nerves to the kidney.

Genito-urinary and rectoanal centers. There are two centers in the lower part of the cord which control the functions of the genito-urinary system and the anal canal. For the sake of simplicity the detailed discussion will be confined to the nervous mechanism controlling the urinary bladder. In the first and second lumbar segments there is a preganglionic cord center whose axones pass through the corresponding lumbar ganglia of the sympathetic and reach the bladder walls by way of the hypogastric plexus. This autonomic mechanism provides for the filling of the bladder. It stimulates the internal sphincter and inhibits the detrusor muscle of the bladder wall, thus relaxing the bladder and allowing it to fill. This cord center is not under voluntary control.

In the second and third, or third and fourth sacral segments of the cord is the pelvic parasympathetic cord center. From it efferent nerves pass by way of the corresponding ventral nerve roots to the vesical plexus, and by way of the internal pudendal nerve to the deep transversus perinei and the sphincter of the membranous urethra. This center provides for the emptying of the bladder; it inhibits the sphincters and stimulates the detrusor urinæ. It is partly under voluntary control. In infants the bladder and bowels empty automatically, but with the development of the pyramidal tracts and prolonged training reflex micturition may be

* These white rami communicants all contain visceral sensory branches from the various abdominal organs which pass through the ganglia uninterrupted.

controlled, and it may be initiated by voluntarily relaxing the sphincters (at least the striated sphincters) and thus inducing a micturition reflex. If a drop of urine finds access to the prostatic urethra active contraction of the detrusor urinæ is set up reflexly and may be aided by action of the abdominal muscles and diaphragm. The detrusor may be voluntarily inhibited, but with difficulty. This, however, is probably reflex inhibition following voluntary activation of the sphincter.

The nervous mechanism for the rectum and anus is similar to that for the bladder. The autonomic center in the first and second lumbar segments, by way of the sympathetic ganglia and inferior mesenteric plexus, maintains the tone of the internal sphincter ani and allows the storage of feces in the lower colon. The parasympathetic center in the third and fourth sacral by way of the internal pudendal nerves acting in response to pressure from within, relaxes the sphincters and thus initiates reflex peristalsis of the colon. This lower center is under control of the will. There are probably two cortical centers for the control of the parasympathetic nuclei in the third and fourth sacral segments. One of these, situated between the shoulder and hip centers in the anterior central gyrus (Fig. 90), controls the voluntary sphincters of the urethra and anus, and the bulbo-cavernosus; the other, situated in the paracentral lobule (Fig. 93), inhibits the sphincters. Probably also there is an infracortical (automatic cord) control, for the bladder and bowels are not troublesome in pure pyramidal lesions, but double pyramidal lesions such as lateral sclerosis may be associated with difficulty in voluntarily initiating or inhibiting micturition.

Cord reflex centers. The cord segments controllng the chief *deep and superficial reflexes* are shown in Figure 95. The more important of these are discussed in the preliminary considerations, Part III (page 184). Figure 95 also shows the *segmental supply* of some of the leading muscles. A full table for the limb muscles will be found at the end of the Classification (page 330). The segmental supply of skin areas is illustrated in Figure 96 and is discussed in Part III, Introduction (page 183).

The initial flaccidity and the return of reflexes which occur in complete cord lesions are dealt with in Part III, Introduction.

SYMPTOMS COMMON TO ALL TRANSVERSE LESIONS OF THE CORD, PARTIAL OR COMPLETE

Transverse lesions of the cord, whether partial or complete, present a series of symptoms (Fig. 109) which are common to all.

(*a*) There is an area corresponding to the lesion where the anterior gray columns are destroyed so that there is consequent flaccid paralysis of the muscles supplied by these segments; owing to the destruction of the lower motor neurons, there is electric reaction of degeneration and subsequent muscular degeneration, if the patient live long enough.

(*b*) A segment above the lesion, there is local congestion or œdema of the cord which gives rise to symptoms of irritation or more probably altered conduction. The result of this is an area of temporary hyperæsthesia above the anæsthetic zone and perhaps muscular tonic spasm from heightened reflex irritability.

(*c*) In the immediate neighborhood of the lesion there is occasionally more or less involvement of the nerve roots, paralytic or irritative. This is extramedullary, as when a nerve root is caught between the fragments of a fractured pedicle, and the symptoms must be differentiated from those due to the cord injury.

(*d*) There is paralysis in muscles below the lesion, at first flaccid if the lesion is complete, spastic if the lesion is incomplete. This paralysis is more or less widespread according to the extent of the injury. During the period of shock the paralysis is flaccid, even in incomplete lesions.

(*e*) More or less anæsthesia or paræsthesia below partial lesions; absolute anæsthesia below complete lesions.

(*f*) In patients with complete division of the cord who live until full establishment of the cord reflexes there may be general flexion spasms, erection, micturition, and profuse sweating (mass reflexes) elicited by pinching the skin anywhere below the lesion, except in a narrow band corresponding to the lesion itself where the spinal reflex arc is interrupted. The width of this band of lost reflexes may afford valuable clinical evidence of the extent of the destruction (*Revue Neurologique,* July, 1922).

The descriptions given below of transverse lesions of the cord should be read with constant reference to Figures 94 to 96.

EXAMPLES

1. A complete transverse lesion at the site of the *third or fourth cervical segment* or above it (second or third cervical spines) causes rapid death by paralysis of the diaphragm and intercostals (Fig. 95) or sudden death by involvement of the vagus nuclei in the oblongata.

2. A complete lesion at the level of the *fifth and sixth cervical segment* (fourth and fifth cervical spines, Fig. 98) causes complete flaccid paralysis of the arms, trunk and legs. The skin over the deltoid and perhaps down

to the nipple in front is hyperæsthetic; this area is supplied by the descending cutaneous branches of the third and fourth cervical nerves. The costal respiration stops and breathing is entirely diaphragmatic. All reflexes below the lesion are lost. Autonomic centers are paralyzed owing to interruption of bulbar control; hence there is miosis from paralysis of the pupillodilator center (see Fig. 94). There is paralytic dilatation of the cutaneous vessels and profuse sweating, owing to paralysis of the vasomotor center in the cord; priapism is present from passive congestion owing to loss of control of the vaso-motors; and there is retention of urine with possibly a paralytic overflow from the over-distended bladder. There is paralysis of the rectum with fecal impaction or incontinence.

A contribution to the Surgery of the Spinal Cord. Thorburn, p. 8, 1889.
Typical posture in complete transverse cord lesion immediately below C. 5.

The loss of vascular tone causes diminished resistance to injury, and there are rapidly forming bed sores. Death occurs in from one to two weeks.

3. A complete lesion at the level of the sixth cervical segment (Fig. above) differs from this in that the arms are abducted by the deltoid (C. 4, 5 and 6), and laterally rotated by the infraspinatus (C. 4 and 5), and the forearms are flexed by the unopposed biceps (C. 4, 5 and 6). These muscles are supplied by segments above the lesion. The zone of anæsthesia extends from a strip down the arm and forearm, including the thumb and all skin postaxial (medial) to this (see Fig. 96); there is hyperæsthesia over the deltoid and down the preaxial (radial) border of the arm and forearm.

4. In a complete lesion at the level of the seventh cervical (Fig. below)

the arm is abducted by the deltoid and supraspinatus, medially rotated by the subscapularis (C. 4, 5 and 6); the forearms are flexed by the biceps (C. 4, 5 and 6). The combination of medial rotation of the arm and flexion of the forearm lays the flexed forearms across the chest. For the anæsthesia consult Figure 96.

5. A complete transverse lesion of the cord at the level of the *eighth cervical and first thoracic segments* (sixth and seventh cervical spines;

Typical posture in complete cord lesion at level of 7th cervical segment. Fracture crushing 7 C. segment and involving roots of C. 7, 8, D. 1. (See page 253.)

see Fig. 98) will show a hyperæsthetic strip down the middle of the front and the back of the arm and forearm (see Fig. 96) which is supplied by the sixth and seventh cervical segments. The arm and forearm muscles may escape, but the short hand muscles and all the trunk and leg muscles will be paralyzed and flaccid. The pupillary and other autonomic centers are affected as in the higher lesions. Death usually occurs in one to three weeks from defective respiration and interference with the cardiac centers in the upper thoracic segments of the cord (compare Figs. 217 and fig. above).

6. A complete lesion in the *mid-thoracic region* (sixth or seventh thoracic spines; eighth or ninth thoracic segments) causes paralysis of the abdominal and leg muscles, a belt of hyperæsthesia which takes the form of a "girdle sensation" halfway between the ensiform and the umbilicus, with complete cutaneous anæsthesia below this, and paralysis of all autonomic and reflex centers below the level of the lesion.

If the bladder is regularly catheterized from the first, and the urine kept aseptic, and if bed sores are prevented by careful nursing, then after an interval of two to three weeks there may be a gradual appearance of spinal reflexes, a return of tone to the bladder with automatic urination and the appearance of mass reflexes (see Part III, Introduction). There is no return of sensation nor of voluntary motion below the lesion. The patient may live for many months, but usually dies within a year.

7. Fractures of the *lower two thoracic and the upper lumbar vertebræ* are the most common fractures of the spine. In these fractures the lumbar enlargement is injured, and the loss of part of the bladder automatic mechanism makes the recovery of bladder reflexes impossible in complete lesions, and death usually occurs in three weeks or thereabout. (Compare Figs. 94 to 96, and 98).

(*a*) When the *upper part of the lumbar enlargement* is destroyed (first and second lumbar segments, tenth thoracic spine; Fig. 98) the iliopsoas and extensor muscles of the thighs are paralyzed and flaccid and undergo degeneration from destruction of anterior gray column cells. The muscles of the legs and feet are flaccid, but do not show reactions of degeneration; the control of the bladder and rectum is lost. There is anæsthesia of the testicle and of the legs below the inguinal ligament. The buttocks may be hyperæsthetic along the outside, as they are here supplied by the iliac branch of the twelfth thoracic.

Below this level any injury or tumor affecting the cord is exceedingly liable to involve the upper part of the cauda equina, so that a lesion involving the cord at the level of the upper sacral segments may involve most of the lumbar nerve roots (see Figure 98), and the symptoms may be largely those of cauda equina injuries.

(*b*) *Lower part of lumbar enlargement* (first and second sacral segments; twelfth thoracic spine). The gastrocnemii and tibial muscles and probably all the muscles below the knee are flaccid and degenerate; there is complete relaxation of the sphincters; there is anæsthesia of the perineum, the back of the thigh and the outside of the leg and outer side of the sole of the foot.

(*c*) *Lower three sacral segments.* There is loss of control of the bladder and rectum, complete paralysis of the sphincters, anæsthesia of the buttock, the back of the scrotum or labia and the perineum.

(*d*) *Injury to the cauda equina* may simulate injury to the lumbar enlargement very closely (consult Fig. 98). An injury at the third lumbar spine may compress the nerves from the fourth lumbar down, simulating a crush of the lower half of the lumbar enlargement (twelfth thoracic spine). Here the seat of the injury may help in diagnosis; or the second or third lumbar nerve may be injured at its point of emergence from the spinal canal, causing a belt of intense pain or of anæsthesia extending along the outside of the thigh and inner side of the leg; the parts below may remain more or less sensitive. In cauda equina lesions the *paralysis is apt to be more widespread than the anæsthesia,* since nerves under compression conduct sensation after they are incapable of conveying motor stimuli, and the anæsthetic distribution may be quite irregular. Nerves that travel farthest suffer most (Fig. 207).

Partial or incomplete cord lesions. When an acute lesion is incomplete, there may be at first complete loss of motion with flaccidity and complete anæsthesia, but as shock passes off subjective pain, numbness, tingling, or muscular cramps may be complained of; or pin prick or deep pressure may be felt. The extent to which this occurs is in exact proportion to the amount of cord which is uninjured. The reflexes return and in two or three weeks are exaggerated. The pain of pin prick or deep pressure on the legs may be referred to the hyperæsthetic zone, traveling unperceived through the cord below the lesion till it reaches the segments above the lesion and there being wrongly referred. (Is this an argument in favor of cord consciousness?) In incomplete lesions the paralyzed limbs, which are at first flaccid, become rigid as the acute stage passes off, and the plantar response, which is absent or flexor during the flaccid period, becomes extensor. This extensor plantar response (Babinski's sign) is usually a very early indication of returning reflexes. The autonomic centers regain tone and in proportion as they recover the patient has a chance of prolonged life.

An illustrative case of a complete crush of the spinal cord is shown in Figure 217 and p. 251 quoted from Dejerine. A boy of 17, an acrobat, dislocated the spine, crushing the seventh cervical segment of his spinal cord which lies opposite the fifth and sixth cervical spines (Fig. 98) and crushing the eighth cervical and first thoracic nerves as they passed through the intervertebral foramina. The figures illustrate his sensory condition

eight months after the injury and the typical position of the arms. He died some time later and the diagnosis was confirmed at autopsy.

Paralysis. There was lower motor neuron paralysis of the muscles supplied by the seventh and eighth cervical and the first thoracic nerves (see Fig. 95) with deformity of the hands like that in Figure 152 or Figure 216 from paralysis, with wasting and reaction of degeneration in all the intrinsic hand muscles and in the long flexors and extensors of the fingers. There was flaccid paralysis of upper motor neuron type of all trunk and lower limb muscles; the diaphragm and serratus anterior escaped. There was paralysis, with flaccidity, but no wasting nor reaction of degeneration of lower limb muscles. There was fecal and urinary incontinence.

Sensory loss (Fig. 217). There was complete loss of all forms of sensation from the distribution of the seventh cervical segment downward (compare Fig. 96). In the skin area of the fifth and sixth cervical segments the sensory loss was of the syringomyelic type, that is, pain and thermic sense were lost, touch sense was retained.

Reflexes. Cutaneous reflexes, abdominal and cremasteric reflexes were normal. The plantar response was flexor. Pinching the skin of the trunk or of the lower limbs produced very active flexion at the hip and knee joints.

Sweating. There was anidrosis of the paralyzed region.

Comment. A crush of the spinal cord such as this often produces a bloodless pulping of the cord within the pial sheath which is not ruptured. The pulpy matter is forced upward and downward within the gray matter, sometimes invading the posterior columns for one or two segments above and below the site of the crush. Hence the syringomyelic symptoms for the distribution of two cord segments above the crush; the second sensory neurons for pain, heat and cold were destroyed as they decussate in these segments, while touch and pressure in this case traveled upward in the posterior columns which escaped.

As has been stated the plantar response in this patient after he recovered from the initial shock should have been extensor in type. The bladder should have acted automatically. Such patients can sometimes induce micturition by scratching or pinching the skin on the inner surface of the thighs. A full bladder in these patients frequently causes vomiting and profuse perspiration of the trunk, which are relieved by emptying the bladder.

It seems remarkable that this patient lived so long, as his sympathetic cardiac nerves must have been paralyzed and he cannot have had costal

respiration but must have depended entirely on his diaphragm and serratus anterior for respiration.

LESION OF ONE LATERAL HALF OF THE SPINAL CORD
(Brown-Sequard Paralysis)

The commoner causes of a lesion of one lateral half of the cord are hematomyelia, either traumatic or spontaneous, tumor of the meninges or cord, stab wound, or concussion by bullet or shrapnel. (See Gordon Holmes, "Cases in the Late War," *British Medical Journal,* Dec. 11, 1915.)

An imaginary typical case is illustrated in Figures 109 and 110, the half lesion affecting the right half of the sixth and seventh thoracic segments of the cord. Figure 109 illustrates the cord as seen from the front. The description is to be read with constant reference to the figures.

Motor symptoms.

(*a*) *Motor Loss for the Segment Involved.*

1. The cells of the anterior gray column are destroyed on one side for the extent of the lesion; this results in lower motor neuron paralysis with flaccidity and degeneration of the muscles or parts of muscles supplied by the sixth and seventh thoracic nerves (sixth and seventh intercostals, upper segments of abdominal muscles).

(*b*) *Motor Tract Symptoms.*

2. The lateral cerebrospinal tract supplying voluntary control to all the muscles below the lesion on the same side of the body is interrupted, so that there is upper motor neuron paralysis of the same side of the body below the lesion, viz., paralysis of the right lower intercostals, the right side of the abdomen, and the right leg. Most recorded cases show spasticity in extension with increased reflexes.

3. Bladder and bowels do not suffer permanently in half lesions of the cord, as unilateral cerebral connections are sufficient for proper control.

4. Bulbar control of the vasomotor centers in the thoracic cord are interrupted, so that there is vascular dilatation and possibly dryness of the skin on the side of the lesion (compare Fig. 94). Owing to the vasodilatation there may be a relative elevation of temperature as compared with the opposite side.

Sensory symptoms.

(*a*) *Root symptoms.*

5. The sixth and seventh nerve roots on the right side are completely interrupted as they pass into the injured segment. There is therefore a

zone of complete anæsthesia on the right side corresponding to the distribution of the sixth and seventh thoracic nerves on the side of the lesion.

6. Referring to Figure 109 one can readily imagine a zone of slight alteration of the cord (hyperæmia and œdema) above, below, and opposite to the area of complete destruction. Through all this area the conduction is not interrupted, but is altered and probably delayed. The right fifth thoracic nerve enters this altered zone of cord above the lesion, and the imperfect conduction through this area accounts for the occurrence of a narrow strip of hyperæsthetic skin above the anæsthetic skin on the side of the injury (Fig. 110).

7. The *right* eighth thoracic nerve (partly), and the right ninth and tenth thoracic nerve roots pass through a similar altered region of the cord below the lesion before they cross; therefore, there is a zone of hyperæsthesia below the anæsthetic strip on the side of the lesion. The hyperæsthetic symptoms are usually transitory.

8. The *left* sixth and seventh or seventh and eighth nerve roots enter the altered segments of cord immediately opposite the lesion. There is, therefore, a zone of hyperæsthesia corresponding to the distribution of these nerves on the left side (the side opposite to the injury). This corresponds in level with the zone of *complete anæsthesia* on the side of the injury (see Fig. 110) and is therefore at a *lower level* than the upper hyperæsthetic area on the injured side.

(*b*) *Sensory tract symptoms.*

9. *The sense of passive position, of active and passive movement, stereognostic sense, tactile discrimination* (*compass sense*), *bone conduction* (*tuning-fork sense*) *and texture* all travel by the homolateral posterior columns. Therefore, all these forms of sensation are lost on the side of the lesion but are not affected on the opposite side.

10. *Sense of touch and pressure.* These forms of sensation travel usually by two paths, one homolaterally in the posterior column, the other in the crossed ventral spinothalamic tract. In Holmes' cases of half lesion of the spinal cord from gunshot injuries (*British Medical Journal,* Dec. 11, 1915) remote (*i.e.,* tract) tactile loss was unusual, but when touch was lost, "there was greater diminution of sensibility to light touch on the side of the lesion."

11. *Tract for pain, heat, and cold.* The lower sensory neurons for pain, heat, and cold enter the posterolateral column of the cord and run upward in this column two to six centimeters before being relayed by the second sensory neuron. The second sensory neuron for pain, heat, and cold crosses in the gray matter and gray commissure to the opposite pos-

terior spinothalamic tract. The height above the level of entry at which the crossing takes place varies in different regions of the cord.

No crossing at all takes place below the twelfth thoracic segment; the lowest sacral nerves cross in the twelfth thoracic segment; this segment is 4 or 5 cm. from the lower end of the conus medullaris. In the mid-thoracic region the crossing for thermal sense is one segment (1½ cm.) above where the nerve enters the cord; the path for pain and touch runs up two segments (3 cm.) in the cord before crossing. In Figures 109 and 110 the difference in the levels for pain, heat, and cold are not indicated. In the upper thoracic region pain and temperature cross two to three segments (3-5 cm.) above the point of entry; at the cervical enlargement three to four segments above the entry (3-5 cm. as the segments are shorter in this situation). At the fourth cervical the crossing is four to five segments higher up (5-6 cm.).

In the spinothalamic tracts the more caudal fibers are more peripherally placed and tend to recover first. (Gordon Holmes, B. M. J., Dec. 11, 1915.)

From these facts it follows that below the twelfth thoracic segment the sense of pain, heat, and cold is lost for the half of the body below the lesion and on the same side. But if the half lesion of the cord occurs above the twelfth thoracic segment, the posterior spinothalamic tract, which carries pain, heat and cold from the opposite side of the body, will have crossed below the lesion, and the sense of pain, heat and cold is lost on the opposite side of the body commencing two or more segments below the level of the lesion (Fig. 109).

12. *Tract hyperæsthesia.* On examining Figure 109 it will be seen that the posterior spinothalamic tract for pain, heat and cold on the side opposite the lesion passes through a part of the cord more or less altered (hyperæmic, edematous) by its contiguity to the injured portion. Therefore, the conduction here is defective and the result is that for a time at least the lower part of the trunk and the leg on the side of the injured cord is slightly hyperæsthetic as well as hypæsthetic. That is, the conduction of pain, heat and cold and sometimes touch or stroking is slightly delayed and the reaction when it passes through the obstruction is accentuated.

13. *Reflexes on the side of the lesion.* The Babinski sign is positive (extensor plantar response) and there is loss of cremasteric and abdominal reflexes, with increased tendon reflexes due to interruption of the pyramidal control.

On the side opposite the lesion the plantar response is flexor (*i.e.*,

Babinski negative), skin reflexes are present, and pain reflexes are absent.

Babinski's surréflectivité hyperalgésique. During the period when there is hyperæsthesia on the side of the lesion, pinching the skin in the hyperæsthetic area on the side of the lesion produces no reaction on the side of the lesion, or at most only the slow dorsiflexion of the ankle characteristic of the defense reflex, but there occurs an abrupt movement of the opposite leg accompanied by quick, noisy inspiration (as of pain) and a very disagreeable sensation. It can even be produced by pinching the skin above the lesion, but again on the side of the lesion, only the pinching must be more severe. Babinski thinks the reaction cortical. He is sure it is not voluntary. (*Revue Neurologique,* 1921, page 434.)

Summing up these symptoms we get the following Brown-Sequard syndrome:

On the Side of the Lesion

(*a*) Lower motor neuron paralysis for the nerve root involved.

(*b*) Upper motor neuron paralysis below the level of the lesion.

(*c*) Complete anæsthesia for the skin corresponding to the half-segment of the cord involved.

(*d*) Loss of sense of position, sense of motion, tuning-fork and compass senses, and stereognosis.

(*e*) Sometimes loss of sense of touch and pressure.

(*f*) A temporary zone of hyperæsthesia above the area of complete anæsthesia.

(*g*) Hyperæsthesia to touch, tickling, pain, heat and cold, best marked immediately below the anæsthetic zone, but extending all down this side. This is usually only temporary.

(*h*) Relative elevation of temperature owing to vasomotor paralysis.

(*i*) Anhidrosis.

(*j*) No loss of sense of pain, heat or cold.

(*k*) Plantar response extensor, skin reflexes lost, tendon reflexes increased.

(*l*) Babinski's surréflectivité hyperalgésique.

On the Side Opposite the Lesion

(*a*) No loss of voluntary movements.

(*b*) A zone of hyperæsthesia corresponding to the cutaneous level of the segment immediately opposite the lesion.

(*c*) Loss of pain, heat and cold for lower half of the body commencing 2 to 6 cms. below the level of the lesion.

(*d*) No loss of sense of position, motion, stereognosis, tuning-fork or compass sense.

(*e*) Loss of tactile sense in a few cases.

(*f*) Plantar response flexor, skin reflexes present, pain reflexes absent.

In tumors frequently and in fractures and gunshot injuries occasionally a nerve root or roots may be involved independently of the cord lesion, and radicular symptoms may be added, usually on the side of the cord lesion. Thus there may be root pain or anæsthesia and paralysis of radicular distribution, somewhat obscuring the level of the lesion.

These symptoms as summarized should be compared with the hypothetical case illustrated by Figure 110, and with the actual case of shrapnel concussion, Figure 214, taken from Stewart's *Symptomatology of Nervous Diseases.*

Figure 213 illustrates a case of partial lesion of one-half the spinal cord by *spontaneous hematomyelia.* The anomalous point in this case is that there is no lower motor neuron paralysis on the side of the lesion, so the anterior gray columns have escaped as well as the nerve roots. The paralysis on the side of the lesion is of the upper motor neuron type, but the hand muscles alone are markedly affected; the leg is only paretic and slightly spastic.

Level of the Lesion Relative to the Spinal Column. As operation is sometimes beneficial in partial lesions of the spinal cord it is important to be able to determine the level of the lesion and the relationship of the affected segment to the spines of the vertebræ. One must remember the varying distances which the roots of the sensory nerves for pain, heat and cold travel upward homolaterally in the posterolateral column before the crossing takes place. Also one must not forget that the spinal nerve roots, motor as well as sensory, have a varying obliquity within the dural sheath before they emerge by the intervertebral formina (see Fig. 98).

Chipault's rule for determining the relation of the cord segments to the spinous processes of the vertebræ is as follows: "In the cervical region add 1 to the number of the vertebral spine and this will give the segment opposite it. In the upper dorsal add 2; in the 6-11 dorsal add 3. The lower part of the eleventh dorsal spine and space below it are opposite the three lower lumbar segments. The twelfth dorsal spine and space below it are opposite the sacral segments." To find the spine corresponding to the involved segment subtract instead of adding.

Cervical region segment	— 1 = spine.
Upper dorsal segment	— 2 = spine.
6 to 11 dorsal segment	— 3 = spine.
3 lower lumbar segments	= 11th dorsal spine and space below it.
Sacral segments	= 12th dorsal spine and space below it.

Simple reference to such a figure as 98 obviates the necessity of all such calculations.

These rules may be applied to a hypothetical case—hypothetical because few cases recorded are typical in all respects; almost none present a perfect half lesion. Figure 110 represents a patient with *paralysis accompanied by increased reflexes* from the ensiform cartilage down on the right side; the left side is about as strong as normal. The figure of the cord (Fig. 109) is viewed from the front. The patient's sixth and seventh intercostal muscles on the *right side are flaccid* and are sucked in on inspiration and feel soft to the touch, while the higher intercostals contract. Below this level the intercostals may be less soft, as the nerve cells supplying them escape injury.

When examined for sensory disturbances, he complains of pain from slight rubbing or scratching or friction of the bedclothes on the right side below a line about halfway between the ensiform and umbilicus. The hyperæsthesia below the lesion on the same side is readily explained by the fact that the left spinothalamic tracts have to pass through an area of congested or edematous cord on their way past the lesion (see Fig. 109). This alters the sensory impulses by modifications in conduction. Further examination shows a narrow area of complete anæsthesia to all forms of sensation running nearly horizontally across the right side of the trunk a little below the ensiform process (just above the hyperæsthetic area); this area corresponds to the nerves passing into the part of the cord destroyed. Above this is a narrow belt of hyperæsthetic skin whose nerves pass through a segment which is injured but still capable of conduction.

On the left side on which there is no paralysis the patient may or may not be sensitive to cotton wool, but he is completely insensitive to all forms of pain and temperature; the analgesia and thermoanæsthesia start about an inch lower down than the anæsthetic belt on the right side. Above this is a narrow hyperæsthetic zone. On the paralyzed right side he cannot discriminate two points of a compass touching him at once from one point, and he cannot feel the vibration of a large tuning fork when the handle is placed against the leg bones, nor can he tell when his eyes are shut how his leg is placed by the physician. These forms of sensation as well as the sense of passive motion, of the position of his leg, and of weight are unaffected on the left side. The paralysis on the right side with loss of pain and temperature sense on the left is typical of a half lesion of the right half of the cord.

What is the level of the lesion? The surest guide is the flaccid paralysis

from the injury to gray matter and nerve roots at the site of the lesion itself. The flaccidity of the sixth and seventh intercostal muscles points to the sixth and seventh nerves or cord segments. Following Chipault's rule for the relation of the spines to nerve segments in the upper dorsal region, the sixth and seventh cord segments should be opposite the fourth and fifth dorsal spines (compare with Fig. 98). Approaching the subject from the less certain upper limit of thermoanæsthesia, the lesion should be in the segment above the first nerve area insensitive to heat and cold, as we know that in this region the nerves conveying heat and cold run up for one segment before crossing. The eighth dorsal nerve area is in our case the first one insensitive to thermal stimuli, and as it runs up one segment before crossing it will refer us to the seventh segment or fifth thoracic spine for the level of the lesion. This would be the level at which to operate should the case seem to require operative interference.

THE EXTRAPYRAMIDAL (INFRACORTICAL) MOTOR MECHANISM

This comprises the cerebellum, the basal ganglia, the nucleus of Luys (nucleus hypothalamicus), the red nucleus, and the substantia nigra.

THE CEREBELLUM

For a summary of the anatomy and physiology of the cerebellum see Part II, page 165. Consult also the short summary of anatomy and symptomatology in the classification (see Supplement).

A condensed account is given here of the relations of the anatomy and physiology of the cerebellum and its afferent and efferent paths to the symptoms of cerebellar disease. This discussion is founded on Sherrington's article on the cerebellum in *Schaefer's Physiology,* Thomas' "Cerebellar Functions," 1912; Dejerine's "Semiologie des Affections du Systeme Nerveux," Stewart and Holmes "Cerebellar Tumors," *Brain,* 1904, and Holmes' "Symptoms of Acute Cerebellar Gunshot Injuries," *Brain,* 1917.

The Functions of the Cerebellum. Sherrington thus summarizes the functions of the cerebellum: "The cerebellum is an organ of a particular class of reactions whose sources are in the sensifacient organs of various senses, especially those with *spatial quality* (vestibular sense, ocular sense of space, and muscle sense). It *supports the tonus* of most, perhaps all, motor root cells, *especially those of eyes muscles,* and *muscles of the neck and spine.* It helps to secure coördinated action of the body muscles for maintenance of attitude and the execution of movements. It is a center

for geotropism and stereotropism. It supports habitual posture, and is importantly concerned with stock and more specialized movements." "It is the head ganglion of the proprioceptive system" (postural sense, vestibular sense, spatial eye sense).

Holmes says: "The cerebellum receives and integrates proprioceptive impulses from all parts of the body, and by virtue of these keeps the motor mechanisms in such a *state of tone* that they can react promptly and efficiently to voluntary impulses and it thus assures the *correct coöperation* of the separate motor centers that are concerned in individual acts."

"The cerebellum '*sets*' or '*tunes*' or regulates the activities of certain motor mechanisms probably spinal, so that the response to a volitional stimulus is *immediate, effective, and proportional* to the intensity of the cerebral impulse."

"All actions are the products of several cortical and subcortical centers, and though it is the cerebral cortex which initiates voluntary movement and probably selects and integrates the impulses for individual acts, the elaboration and coördination of the numerous factors that are concerned in each must depend largely on subcortical centers. If the cerebellum is the head ganglion of the proprioceptive system it is natural that this coördination should be largely done by it."

"It is probable that each half of the cerebellum exerts a *reinforcing action on the eye nuclei for deviation to its own side.* The cerebellar adjuvant action may be due to the synthesis of proprioceptive impressions from eye and neck muscles and the labyrinth."

All recent observers are agreed that the cerebellum is *not represented in consciousness.*

Among cases of cerebellar injuries in wounded soldiers Holmes has never found disturbance of any form of sensation. The patient always has accurate knowledge of the position of his limbs in space. His difficulty in estimating weight may be due to the asthenia of the affected limb, as the weight in the affected hand is estimated as heavier than it is. In none of Holmes' cases was there any evidence of localization in the cerebellum. Small and superficial lesions produced only transient symptoms; these were generalized over the homolateral side of the body, never limited to one limb or to one segment of a limb. However, *in vermis injuries* the head, neck, and trunk muscles, and phonation and articulation, were most affected; therefore, the vermis may be most concerned

with the movements requiring the coöperation of bilateral muscles. Holmes does not believe the cerebellum to be especially an organ of equilibration.

According to Sherrington the cerebellar influence may be exerted: (*a*) directly on the lower motor neuron cells, (*b*) on the thalamus and through its thalamic influence on the cerebral cortex or the efferent basal ganglia. Sherrington especially tends to emphasize the cerebellar action on the infracortical motor apparatus through the thalamus.

In extensive slowly advancing cerebellar lesions great compensation is possible through the opposite voluntary motor cortex of the cerebrum, and in limited acute lesions this mechanism rapidly leads to complete compensation.

Symptomatology of acute cerebellar lesions. It will be observed that all except the grosser symptoms must be carefully searched for by special tests, and that all the tests are practically dependent on the integrity of the pyramidal system for the side of the body involved, so that cerebellar symptoms are largely masked if there be simultaneous pyramidal lesions affecting the same side of the body. It will also be realized that in a very sick patient it may be quite impossible to diagnose a secondary cerebellar lesion. Thus many large cerebellar abscesses and secondary tumors completely escape detection during the life of the patient.

Vermis symptoms are bilateral in proportion as the lesion approaches the middle line; with this exception all focal cerebellar symptoms are overwhelmingly homolateral.

TRANSITORY SYMPTOMS

Diaschisis or shock is never a prominent symptom either in acute lesions of the cerebellum or after cerebellar operations.

Skew deviation of the eyes. In dogs after unilateral ablation of a cerebellar hemisphere the homolateral eye is turned downward and inward, the opposite eye upward and outward (Fig. 125). This is apparently asthenic, as the more permanent effect of a unilateral lesion is weakness in turning both eyes toward the injured side. Skew deviation was present in 12% of Holmes' cases (*Brain*, 1917) lasting for a week or ten days. It is common after cerebellar operations.

Nystagmus. Spontaneous nystagmus when the eyes are not fixed is only present for a short time after operation. Fixation nystagmus is more permanent and will be considered later.

Vertigo consists of a subjective sensation either of oneself turning round or of external objects turning round. Very rarely the patient may seem to be falling backward or forward, or external objects appear to rise or fall before him. Still more rarely external objects may appear to advance or to recede.

Vertigo is present in disease of the labyrinth or vestibular nerve, in cerebellar affections, in weakness of the eye muscles, in sudden cerebral anæmia, or hyperæmia. In cerebellar injuries it is present for the first two or three days only and there is no constant relation between the side of the lesion and the direction of rotation. In intracerebellar tumors it is a rather constant symptom and both the apparent whirling of *objects outside* and the *patient's subjective sense of rotation* are *from the side of the lesion.* In extracerebellar tumors (most of which involve the vestibular nerve) it is a very constant symptom. In this instance the sense of rotation of surrounding objects is *from* the side of the lesion while the patient's own sense of rotation is *toward* the side of the tumor, and this is a very important help in diagnosis.

Occasionally the vertigo is accompanied by *forced rotatory movements.* This is a very constant symptom in dogs after unilateral cerebellar ablation (Fig. 122). The dog falls to the operated side and continues to roll as he falls. These rotatory movements are rare in man, but patients show a tendency to lie on the side of the lesion. (A tendency to lie on the healthy side after operation is noted by Stewart and Holmes. Statements of different authorities do not agree.)

Attitude—Head. In dogs (Fig. 125) with unilateral ablation the homolateral shoulder is advanced ventrally, the occiput is turned toward the side of the lesion, the chin to the opposite side. This is the usual attitude in patients after acute injuries and after operation, but the tendency is less marked than in dogs. It is rather common in cerebellar tumors, but is not pathognomonic. Rarely the position may be reversed, and the same attitude may be noticed in pontine, mid- and fore-brain tumors. In cerebellopontine angle tumors the ear may be tilted toward the shoulder, away from the tumor, but this is not constant (Purves Stewart).

Trunk. As in dogs (Fig. 123) so in man the trunk may be bent with the concavity toward the lesion. In vermis affections the head and trunk may be tilted backward or forward.

These symptoms are transitory in wounds, more constant in tumors of the cerebellum.

THE MORE LASTING CEREBELLAR SYMPTOMS. Figs. 128 A to 132

I. Atonia or hypotonia. One of the earliest, most constant and most persistent effects of cerebellar lesions is homolateral diminution of tone in all voluntary muscles. In hemisphere lesions this hypotonia is best marked in the limbs, less so in the trunk, which depends mainly on the vermis. In limited acute lesions of a hemisphere the tone is largely regained after the first week. In very limited lesions the loss of tone is slight but is generalized. Apparently, however, the rest of the hemisphere which is spared compensates for the loss. The hypotonia is general and uniform and does not affect one group of muscles more than another. The muscles feel flabby to the examiner. The patient long after the acute lesion complains that his affected limbs "feel floppier" than the well limbs. If the elbow be supported and flexed, and the forearm be shaken by the physician, the hand swings in a flail-like manner. The affected limbs are left lying in unusual relaxed attitudes. But there is no relaxation of the ligaments as in tabes. Hypotonia diminishes after wounds, but the muscles never become normal. Figs. 128 A, B.

II. Disturbances of voluntary movements.

(*a*) *Asthenia.* There is no loss of static strength, that is, the fixed arm or leg on the affected side may not be more easily moved by the physician against the patient's resistance than normal, but the strength for active movement as tested by a dynamometer or by the effort to grasp the physician's hand is diminished. The affected limbs tire more quickly. There are sudden unexpected relaxations of effort as of the leg in walking. If the patient is asked to hold both arms out horizontally the affected arm tires quickly. The patient shows an apparent reluctance to use the affected arm. If the right arm be affected he takes his food with the left.

(*b*) There is *slowness* both in starting and in stopping a movement; the affected limb lags behind the other.

This combined asthenia and slowness has been *wrongly called paresis.* It is distinguishable from the paresis of a mild pyramidal lesion by being (1) on the same side as the lesion, while cerebral hemiparesis is always crossed; (2) in the cerebellar affection all muscles are affected equally, while in cerebral paresis the flexors of the leg and thigh and the distal muscles of the upper limb are most affected; (3) in cerebellar affections there is no limit in the range of movements; (4) the reflexes are normal; and (5) there is no rigidity nor contracture.

(*c*) There is *ataxia* of the cerebellar type of the affected side. Broadly,

cerebellar ataxia is a *drunkenness,* which in one-sided lesions affects the homolateral limbs. The leg is spread widely so as to enlarge the base of support; it is lifted too high and set down too forcibly and the patient reels toward the affected side (Fig. 129). The affected arm has the intention tremor of drunkenness, is used awkwardly, jerkily, overshooting the mark (Fig. 130). The speech in vermis lesions is drunken. So much do cerebellar patients suggest drunkenness that it appears that alcohol has a special affinity for the cerebellum.

Passing from this general description, which is most applicable to gross lesions, to particulars, one notices that all movements are somewhat affected, but in disease of the cerebellar hemispheres the arms are more affected than the legs, while in disease of the vermis the ataxia is greater in the legs than in the arms. Complex movements are more affected than simple movements; movements involving several joints more than those involving one joint; rapid and alternating movements more than slow continuous movements; and the distal joints more than the proximal joints. If a patient after a recent severe lesion of the right hemisphere be asked to touch his nose with his left hand, he does it accurately and at once, but when asked to touch his nose with his right hand his arm sways aimlessly, he shoots it out first, then bends it at the elbow, shoots jerkily past his nose and hits his cheek. He complains that the hand "will not go straight," he cannot "get the right direction," he cannot "pull it up quickly enough." Doing it slowly he succeeds better, and he manages complicated spontaneous movements more directly, but they are accompanied by tremors of pronation and supination before the hand comes to rest.

Analysis of Cerebellar Ataxia. Holmes analyzes cerebellar ataxia into five elements, (1) decomposition of movement, (2) asynergia, (3) dysmetria, (4) tremor, (5) deviation from the line of movement. Each of these will be discussed separately.

1. *Decomposition of movement* (Babinski's asynergia). If the patient while standing is asked to put his foot on a chair, instead of doing so with a harmonious, more or less graceful blending of all the necessary movements, he jerkily flexes his thigh first, then his knee, and attains his purpose in an awkward, disjointed manner. His muscles do not work harmoniously. The various components of the movement are not merged one into the other.

(2) *Asynergia* (Holmes). Figs. 128 C to G. The immediate and accessory muscles, the agonists and antagonists and synergists do not act

together. Every movement is normally brought about by the simultaneous action of many muscles. When flexors act the opposed extensors must relax; and in all the more complex movements several joints must act together. Asynergy means the failure of this synchronism which seems to depend largely on a cerebellar coördinating influence. If the patient be asked to flex and extend his fingers alternately the extensors often overact. When he attempts to touch the tips of the fingers of the affected hand in order, with the thumb of the same hand, the fingers flex together, not separately and consecutively. Directed to look toward the affected side, he does not move the head and eyes together. If while he is lying in bed or on the floor he is directed to fold his arms and raise his body, the heel on the affected side often rises from the bed. In getting up from a sitting position he often falls backward, owing to failure to bring his trunk forward to a position of equilibrium in synchronism with his leg movements. When walking the patient's affected arm does not swing in its natural synchronism with the opposite leg (Fig. 131).

3. *Dysmetria.* This means difficulty in exactly measuring the range and force of individual muscular action so as to produce perfect adaptation to the purpose of the movement. It is usually a hypermetria, a shooting past the mark (Fig. 128). Asked to touch his nose with the affected hand the patient goes past his nose and hits his opposite cheek or eye; he complains that he cannot stop soon enough. If he is asked to take a glass of water, drink, and put down the glass, he opens his hand too widely both in taking and releasing the glass (Fig. 129). Dysmetria in the leg may be tested by asking the patient in bed to touch the knee of the well leg with the heel of the affected leg. He bends his hip and knee too far, touches his thigh, and then slides his heel down to the knee. He is perfectly aware of his error and if asked to repeat the movement several times in succession, he soon learns to correct it by cerebral compensation.

4. *Tremor* is most marked in the late stages of cerebellar disease. It is a coarse intention tremor and is best marked when the patient reaches out to touch an object at some distance. It is well shown in the handwriting (Fig. 132), and especially in vermis lesions may appear in lateral or anteroposterior swaying of the head.

When the hands are held out in front, the affected hand may at first be unnaturally steady, steadier in fact than the well arm; this unnatural steadiness is called *cerebellar catalepsy.* But as the patient tires there is an asthenic slow failure and jerky recovery which amounts to a coarse

tremor. Cerebellar catalepsy and tremor are well seen in the leg. The patient, lying on his back, is told to hold both legs in the air. The unaffected leg may take longer to come to rest, but when it has done so it is more motionless than the well leg for a time. Then it droops weakly and is jerkily restored, giving rise to a coarse static tremor.

The tremor of multiple sclerosis may be due to involvement of cerebellar tracts or peduncles.

5. *Deviation* in walking occurs in cerebellar disease. When asked to walk in a straight line the patient deviates at first toward the side of the lesion. Walking round a table which is placed on his affected side he tends to run into the table. If the table is on the well side he tends to circle farther and farther from the table. Soon he corrects this tendency and then he is apt to *overcorrect* it, so that his later tendency is to deviate from the side of the lesion.

SOME SPECIAL SYMPTOMS

The rebound phenomenon. If the elbow be flexed toward the face against the pull of the physician, and suddenly released, in a healthy man the forearm will be arrested automatically before the hand strikes the face, possibly with an extensor rebound. In a spastic limb the rebound may be exaggerated. On the other hand in a cerebellar lesion the flexion movement is continued violently after release till the face is hit. If this maneuver is repeated often the patient learns to control the limb. If the elbow be supported and the patient rapidly flexes and extends the forearm, the range of movement is often excessive. This is always most pronounced in the early stages after injuries of the cerebellum.

Adiadochokinesis (Babinski), or dysdiadochokinesis means difficulty experienced by patients with cerebellar lesions in performing reversed movements in rapid succession, such as making and breaking a fist, pronating and supinating the forearm, flexing and extending the fingers, shaking a pepper duster, shaking hands rapidly, clapping hands, rubbing out with an india rubber, or making the scissors movement, or the to and fro movement with a pen. It is a combination of the defects already mentioned, slowness of starting and stopping a movement, dysmetria, dyssynergia, and difficulty at the turn. It is seen in all joints, especially in the arm, and is a very persistent symptom. (Fig. 132.)

Spontaneous deviation of the affected arm (Holmes) and Bárány's pointing test. In Holmes' test the patient extends the upper limbs sym-

metrically in front of him and the physician steadies them by his palm against the fingers; the patient then closes his eyes, and when the physician removes his steadying hand, the arm on the side of the lesion swings slowly or abruptly away from the position of symmetry and gradually comes to rest. In *Bárány's pointing test* the subject with fully extended arm touches a fixed object, closes his eyes, lets the arm drop and immediately tries to touch the same object again. A normal individual can succeed in doing so, but in cerebellar disease the patient misses the mark with the homolateral limb, which deviates outward and overshoots the mark.

Abnormal attitudes (Figs. 122 to 125). In dogs after ablation of one cerebellar hemisphere the occiput is turned toward the homolateral shoulder, the muzzle toward the normal side; the body is concave toward the operated side; the eyes show skew deviation, that on the operated side turns downward and inward, the opposite eye, upward and outward. The animal rotates round its own axis, the homolateral shoulder passing ventralward, then medialward and so round. In man, after a severe injury to one cerebellar hemisphere, or after operation, there is less tendency to abnormal attitudes or to forced movements, but so far as they exist they are similar to those described in dogs. Where the vermis is injured the face is stolid and expressionless. The flaccid hypotonic limbs lie where they fall, especially those on the wounded side. Recovery from all this is rapid.

Standing and gait. Holmes says that when he first gets up a man with a cerebellar wound, if he tries to walk on all fours, does so peculiarly like an experimental dog (Figs. 124, 125 and 126). When he gets out of bed he is shaky, unsteady in standing, and sways irregularly. His head oscillates; he feels in danger of falling toward the side of the wound and backward; he says he feels pulled over; but he can generally maintain his equilibrium. Later he has difficulty in attaining his balance *when he gets up from a sitting position.*

The attitude in standing is striking. His head and trunk are both inclined to the injured side, the trunk concave toward it (compare Figs. 125 and 129). The pelvis is so tilted that the weight falls on the opposite leg. The homolateral shoulder is generally higher and usually in front of the other, the homolateral leg abducted and rotated outward (Figs. 125 and 129), the whole body is stiff and rigid. The patient can stand as securely with his eyes closed as with them open, but there is great danger

of his falling if his attention be diverted, as he is maintaining his balance by a voluntary effort. This is an example of cerebral compensation for cerebellar defect.

Gait. The patient has obvious difficulty in maintaining his equilibrium (Fig. 129). He tends to fall (feels pulled over) to the affected side, and evidently fears trusting himself on that leg. The affected leg is abducted and rotated outward. It may be dragged, but is more often raised unnaturally high and is then put down forcibly. In walking the patient deviates toward the affected side, but soon notices this and tends to overcorrect it. When walking he has difficulty in stopping suddenly. He cannot synergize his muscles quickly for this purpose. The affected arm hangs inertly by the side, owing to the asynergia (Fig. 131), and the patient does not synchronize his arms and legs in any effort to regain his balance if he feels likely to fall. If pushed he falls stiffly and rigidly like a doll.

Late nystagmus. While the early spontaneous nystagmus soon passes off, oscillation of the eyes when they are turned as far as possible toward the side of the lesion is very constant and persisting. This is an asthenic nystagmus. After a right-sided lesion the patient on directing his eyes toward the right has difficulty in maintaining them in that position. They swing slowly to the left from failing muscular effort and are jerked back again by quick short jerks, only to slowly fail again. This is a lasting symptom.

(Labyrinthine nystagmus is different. It persists only for two or three days after the injury to the labyrinth. It is diminished by an effort at fixation. The slow phase is always toward the injured side whether the eyes are at rest or are deviated voluntarily toward either side.)

Speech. After recent and severe injuries, especially of the vermis, the speech is usually slow, drawling, monotonous, tends to be staccato or scanning, singsong, and difficult to understand. Phonation may be irregular, jerky, and at the end of a sentence explosive. There is apparent effort, accompanied by facial contortions. This is another instance of difficulty in synergizing the muscles.

Reflexes. Skin reflexes are all normal. The tendon reflexes are irregularly affected. The knee jerk is apt to have a pendulum-like swaying character. If the patient be in bed, with the knees slightly bent and propped on pillows, and the patellar tendon is struck, the healthy leg is projected forward and immediately drawn back to its original position. In cerebellar disease the leg on the affected side is projected forward, but

is not drawn back. This is a variant of the rebound phenomenon and is due to a lack of synergic action of antagonistic muscles. This symptom is very persistent.

In *vermis lesions* phonation and trunk symptoms are worse. In *bilateral lesions* symptoms are of the same character but are bilateral. Speech is much affected. Cases of *pure cortical lesions* recover rapidly from most of the more apparent symptoms. Nuclear lesions take much longer to recover.

The symptoms and tests of cerebellar disease have been epitomized from Holmes thus fully because most textbooks pass over this subject very shortly, and I have found that students have great difficulty in knowing how to examine patients for cerebellar symptoms. In applying the tests to the chronic cases more commonly met with in civil practice one has to remember that the more chronic the condition the greater the opportunity for compensation; but a certain amount of atonia, asthenia, asynergia, discontinuity of movement, asthenic nystagmus on conjugate deviation of the eyes toward the lesion, and asthenic intention tremor can be found by careful examination in most cerebellar cases. The student can now fully understand that most of these symptoms cannot be searched for in the presence of any pyramidal affection of the suspected limbs. Further, it must be remembered that cerebellar symptoms may be due to a lesion of the cerebellar cortex, the cerebellar nuclei, or any of its afferent or efferent paths; and only the history of the case and collateral symptoms may determine the site of the lesion. If the student will now consider the cerebellar affections as they are listed in the classification, he will be able to adapt the list of symptoms to particular cases. (See Supplement.)

THE BASAL GANGLIA

Corpus striatum. The function of the corpus striatum is entirely motor (Fig. 87, a, b and f). It consists of two parts, the palæostriatum or ancient striate body which in mammals is the globus pallidus, and the neostriatum or new striate body, including the putamen and caudate nucleus. The palæostriatum alone is found in fishes, and in mammalia, including man, this develops earliest; the neostriatum appears first in amphibia and develops later; it is found in all vertebrates above fishes.

In fishes the palæostriatum is the highest cerebral motor center, in birds the neostriatum appears as a higher controlling mechanism, acting probably over and through the palæostriatum; there is no pyramidal

system. In mammalia there first appears a cortical motor (pyramidal) system which reaches its highest development in man; the striate motor system remains and takes on some lower function not yet well understood. In a dog the striate body with the thalamus and coördinating action of the cerebellum may enable it to walk and eat and perform the usual automatic actions of the body in the absence of the cerebral cortex, but this is not possible in monkeys and man.

The caudate nucleus receives afferent stimuli from the optic thalamus. These it conveys to the putamen, which in turn acts on the globus pallidus (Fig. 87, f). The globus pallidus is rich in large cells of efferent type. It sends fibers to the thalamus by which it may possibly control thalamo-cortical reactions on the upper motor neuron; and it reaches the lower motor neuron by a relay in the red nucleus, by way of the rubrospinal tract and by relays in the nucleus hypothalamicus and substantia nigra which perhaps reach the spinal cord by axons intermingled with the pyramidal tracts.

Physiology. Experimentally the globus pallidus appears to exert in monkeys a steadying influence on the contralateral limbs which on anatomical grounds may be exerted (*a*) on the cerebral cortex through the optic thalamus, (*b*) on the lower motor neuron through the red nucleus, corpus subthalamicus and the substantia nigra. There is no evidence (at least in monkeys) of cortical control over the striate body.

See Anat. and Phys. of Corp. Striat., Wilson, *Brain,* 1913-'14; for a late résumé of the subject see the *Revue Neurologique,* 1921, page 534; also S. A. K. Wilson, *Lancet,* Aug. 8, 1925.)

Clinical Phenomena. In *Brain,* XXXIV (1911-'12), Wilson, under the heading, "Progressive Lenticular Degeneration," published a series of cases characterized pathologically by a bilateral symmetrical softening in the lenticular nucleus involving more particularly the putamen; the globus pallidus to a less extent. In a pure case the internal capsule entirely escaped. Secondary degenerations affected (and demonstrated) lenticulo-thalamic and especially lenticulo-rubral fibers. No other part of the brain was diseased. There was also a profound cirrhosis of the liver, but this gave rise to no symptoms.

The symptoms of this purely lenticular lesion are thus summarized by him and form a syndrome of the corpus striatum:

"The symptom complex (see Figs. 133 to 136) consists of involuntary movements, nearly always a bilateral tremor of both upper and lower extremities, the head and trunk also being sometimes involved, the tremor

usually rhythmical but occasionally irregular, increasing with volitional movement; pronounced spasticity of the limbs and face, the latter usually set in a spastic smile; in later stages contractures of the limbs. There is dysphagia (difficulty in swallowing) and dysarthria (difficulty in articulation), later complete anarthria (inability to speak from motor defect, here from motor spasm), sometimes spasmodic laughing and emotionalism. The rigidity causes difficulty in standing. There is no true paralysis or paresis, most ordinary movements being executed, but slowly and feebly owing not to pyramidal defect but to muscular rigidity. Sometimes transitory mental symptoms. Abdominal reflexes unaltered, plantar response flexor. Death occurs in a few months to four years. It affects young people and is often familial."

These cases appear to prove that the lenticular nucleus exercises a "steadying effect" on the cortico-spinal system (pyramidal pathway) either directly through the lenticulo-rubrospinal path, or by unknown tracts probably blended with the pyramidal fibers from the nucleus hypothalamicus and substantia nigra, or indirectly through a lenticulo-thalamo-cortical path. The clinical fact that lenticular degeneration is always associated with general spasticity of all the voluntary muscles (the eyes only escaping) seems to the writer to explain why a purely cortical paralysis is very mildly spastic, while a paralysis from capsular hemorrhage in which the lenticular nucleus and lenticulo-rubral fibers suffer along with the pyramidal tract is always markedly spastic.

Since Wilson's article appeared many other cases of progressive muscular rigidity with tremors have been reported, some of them with complete autopsy findings. A group of these seems to be separable as distinguished from Wilson's cases by being more slowly progressive, of no family tendency, without hepatic cirrhosis, not incompatible with long life, and showing a slow disappearance of the cells of the globus pallidus without softening. (Ramsay Hunt, *Brain,* 1917.)

Paralysis agitans is a disease of late middle life characterized by progressive rigidity, coarse tremors, characteristic mask-like countenance, loss of automatic movements, bent rigid attitude, and festinating gait, monotonous explosive speech, little sensory disturbance, and no tendency to fatality. This symptom complex suggests disease of the basal ganglia, red nucleus, nucleus of Luys or substantia nigra as its probable cause, and recent pathological findings are pointing this way. (Fig. 137.)

Huntingdon's Chorea and Athetosis (Figs. 200 and 201), diseases in which involuntary movements are the chief characteristics, have been

associated with cellular degeneration in the neostriate bodies (Mme. Vogt).

The *chorea of children* and of *pregnancy* (Sydenham's) appear to be due to some toxin or toxins affecting the neostriatum.

The recent epidemic of encephalitis lethargica has furnished a great many cases where the inflammatory process characteristic of this affection has been concentrated in the thalamus and basal ganglia; the *Revue Neurologique* for 1921 is very rich in cases with autopsy findings bearing on this subject. In proportion as rigidity predominates as in Paralysis Agitans and in the Parkinsonian type of lethargic encephalitis, the globus pallidus and substantia nigra are most affected. The choreiform types are characterized by changes in the caudate nucleus and putamen and types showing athetosis and torsion spasm present simultaneous affections of the neostriatum and cerebral cortex.

THE CEREBRAL CORTEX

The symptomatology of disease of certain areas of the cerebral cortex has been considered. It remains to discuss it as a whole.

If one examines a picture of the cells of the cerebral cortex, preferably of the anterior central convolution in the motor or large pyramidal cell area, stained by Golgi's method to show cells and fibers, one is struck by certain outstanding features (Fig. 27; also figure in any physiology).

The afferent fibers ramify almost exclusively in the two superficial layers. The large pyramidal cells which alone send their axons to the motor nuclei of the brain stem and cord (to the lower motor neurons) are not only confined to the limited motor area of the cortex, but are few in number even there (Figs. 27, 90, 91, 140 and 141). All the other cells and fibers of the cortex are thus to an overwhelming extent concerned in the work of association (Fig. 149). Thus all cerebral activity is the sum of many impressions from many sources resulting at last in certain motor results which are partly active and partly inhibitory.

The measure of a man's mentality depends on his large receptivity, wealth of stored cell memories, and powers of choice of many possible reactions; his sanity depends on his powers of balancing and discriminating between received impressions, and regulating resulting reactions. This will depend on the wealth and integrity of his cortical cells and the paths of greater and less resistance created by habit. Bolton appears to have established that amentia, congenital idiocy, is anatomically characterized by *poor development* of the association cells of the whole cortex, especially

the small pyramidal cells of the layer next to the surface layer (Fig. 27), and that dementia (insanity) is characterized by *degeneration* of these cells. As a rule neither case presents any gross lesion.

Let us recall certain anatomical and physiological facts. Comparing the figures, 138 to 145, the central (Rolandic) sulcus, Figure 138, separates the anterior central from the posterior central convolution. It ends on the medial surface of the hemisphere pointing backward and downward a little in front (Fig. 139) of the upturned end of the sulcus cinguli. The area which alone contains giant pyramidal cells of Betz and which therefore alone contributes to the pyramidal tract, occupies part only of the anterior central convolution (Figs. 90 and 140) and includes the anterior wall of the central fissure (Fig. 91). Of this area the lower two-fifths controls the head, the next two-fifths the arm, and the upper one-fifth, with a small portion of the medial surface controls the leg and the perineal muscles (Figs. 90, 140, 142 and 143). Horsley did not believe that it had been proved that the lower anterior central area (the head area) does not send cortical fibers to nuclei of motor cranial nerves, though this area contains no giant pyramidal cells. The short distance to the cranial motor nuclei, he thought, might account for the smaller size of the largest pyramidal cells here.

Various combinations of movements leading to results of a volitional character, and not contractions of individual muscles, are represented in the cerebral cortex. Stock movements, such as sitting, standing, walking, equilibration, may possibly be largely taken care of by the thalamus and basal ganglia with the coördinating action of the cerebellum; but in monkeys and man these require at least a certain amount of cortical (pyramidal) control.

Usually the central sulcus presents an upper and a lower knee, each with the convexity forward, with a receding angle between them. At the level of the upper knee is the center for the hip, at the lower knee the thumb is represented; shoulder movements are elicited by stimulating the anterior central gyrus at the level of the receding angle. In any individual case, however, requiring accurate localization of centers before ablation, preliminary stimulation with minimal electric stimuli is necessary. Horsley has shown in man that surgical ablation of the cortex for the arm causes immediate flaccid paralysis of the arm, with considerable loss of sensation, especially affecting the sense of position and of passive movement and stereognosis. One year after, there was in Horsley's case slight stiffness of the little finger, some return of voluntary movements

(Horsley thought by a posterior-central-thalamo-rubrospinal path), complete astereognosis, and slight tactile anæsthesia of the ulnar border of the hand (Brit. Med. Jour., 1909, Vol. 2). The conclusions are that purely cortical destructive lesions of the motor area cause paralysis with some anæsthesia, especially of muscle sense, and with little rigidity.

The cortical type of paralysis is monoplegic; one limb is affected, or combinations of neighboring areas as the head and the fingers, the shoulder and the hip. One lesion never affects the head and the leg, leaving the arm unaffected. Though this patient of Horsley's had marked astereognosis, other purely cortical lesions have demonstrated that when the anterior central convolution alone is involved, there may be no loss of any form of sensation.

The posterior central convolution is mainly sensory; it is the main end station of the thalamo-cortical fibers. It and perhaps the area of the supramarginal and superior parietal convolution near the horizontal limb of the post-central sulcus (interparietal sulcus, Figs. 140, 142) store the sensory memories underlying volutional movement, the sense of position of the limbs, the sense of passive motion, tactile discrimination, the location of the point of contact called topognosis, and stereognosis. Simple touch, pressure, pain, heat, and cold have their end stations in the thalamus, but are stored as memories in the cerebral cortex.

Destructive lesions of the posterior central cortex when the period of shock has passed do not cause loss of the elementary sensations of touch, pain, heat, and cold; all these reach consciousness in the thalamus. Lesions of the posterior central gyrus cause errors of judgment in postural sense, recognition of the spot touched (topognosis), widening of the distance between points in the compass test, and difficulty in recognizing the shape of flat objects. As the hands and feet are the most highly educated tactile and postural organs, they suffer most. The tactile recognition of familiar solid objects suffers severely. Where the parts of the parietal lobe along the lateral sulcus are affected, errors in judgment of degree of temperature, and textural judgments are most affected (the sense of more-or-lessness). For more full treatment see Figures 140, 221 and 222.

As a result of defective reception of muscle sense there may be more or less cortical ataxia; the patient may have difficulty in performing coördinated movements (buttoning his collar, *e.g.*), without the aid of sight. Here a distinction must be made between cortical ataxia which is one-sided and usually affects one limb only, and cord ataxia (such as locomotor ataxia) which is bilateral.

Cortical lesions may be not destructive, but irritative, as when a spicule of bone from a depressed fracture causes convulsions (Jacksonian epilepsy). These symptoms are usually not continuous, but periodic, with a tendency to recur at shorter intervals. The initial twitching indicates the site of the trouble if it is near the motor area; the irritation tends to spread in regular progression over the whole hemisphere and even to the opposite side, as the ripples spread eccentrically in a pool when a stone is thrown into it.

Thus a spasmodic twitching of the thumb, followed by gradual spread of convulsions to the forearm and neck, shoulder and head, and then to the trunk and legs, would indicate irritation in the neighborhood of the lower genu of the central sulcus (see Figs. 90 and 142). The initial irritation may be sensory. A tumor near the olfactory area (Fig. 143) may cause generalized convulsive seizures preceded by an initial sense of offensive (rarely pleasant) odor—an olfactory aura.

Again, a tumor destructive of one center may cause periodic irritation of neighboring centers. Thus a tumor in the posterior central gyrus, causing loss of muscle sense in the hand or arm and astereognosis, may cause periodic convulsions commencing in the hand or arm; or a tumor in the left superior temporal convolution, causing psychic deafness, may give rise to periodic convulsions starting in the right side of the face; or a lesion causing paralysis of the arm may cause periodic convulsions commencing in the head, neck and leg. I must again reiterate that all cerebral symptoms are crossed; the left hemisphere controls the right side of the body.

Acquaintance with the anatomy and physiology of the cerebral cortex in higher apes, coupled with rapidly accumulating evidence in human patients where careful records of symptoms have been followed by complete pathological reports after death, shows marked correspondence between Campbell's areas (Figs. 140 and 141) and cortical function as it is being worked out in man.

In front of the limited area occupied by giant pyramidal cells (Figs. 140 and 141) which alone gives origin to the cerebrospinal (voluntary motor) tract lies a large area (Fig. 140) of allied structure, but without giant cells. This area apparently stores memories derived from previous experience which underlie skilled movements. Influenced by the will it is the first stage of departure for nerve stimuli which act on the giant pyramidal cells by way of short association fibers and through them produce skilled movements.

Of this large area the inferior frontal convolution in the left hemisphere in right-handed people (Fig. 142) apparently stores motor memories for speech. The posterior ends of the superior and middle frontal gyri, including part of the medial surface, stores motor memories which can be called forth by the will to originate skilled movements of the hand and arm (Fig. 142). There is evidence that while these motor memories for the arm may be stored in both hemispheres, they can only be released by the will, or made available for willed actions as contrasted with cerebral reflexes, through the mediation of the left hemisphere in right-handed people (the right hemisphere in the left-handed). This means that in the right-handed the left hand also can be reached *by the will* only through the left hemisphere by means of fibers reaching the right cortex by way of the corpus callosum. This matter will be more fully discussed under the heading of apraxia (see Figs. 142 and 173).

So also while the area immediately behind the central sulcus is in direct relation with the thalamic afferent fibers, and is therefore the immediate sensory area of the cortex for cutaneous and deep sense, the adjoining portions of the posterior central and supramarginal and superior parietal convolutions store up sensory memories of contact, form, weight, and consistency (hardness, softness, roughness, etc.). These memories are the basis for comparing present with previous experiences and so forming sensory judgments of form, weight and consistency.

Similarly, a small area in the superior temporal convolution is the immediate receiving station for auditory impressions (Fig. 142). Each ear is represented in both hemispheres for simple sound. There is, however, a considerable area in the left superior temporal convolution which in the right-handed stores auditory memories, and this again can be divided into an area storing word memories and another storing musical memories (Figs. 90 and 92).

In the cortex adjoining the calcarine fissure is situated the immediate end station for the reception of visual impressions for the corresponding half of each retina (Fig. 143). For example, the left calcarine area (area striata) receives visual impressions from the left half of each retina or the right half of the field of vision (Fig. 105); the object of direct attention, however, is represented in both hemispheres; that is, the fovea centralis of each retina seems to have bilateral representation, though this is probably not the case in all individuals. Besides this vislal end station there is a large area in the occipital cortex on both the medial and lateral surfaces of the hemispheres which is concerned in storing up visual memories and

in interpreting visual images in terms of previous experiences (Figs. 142 and 143). Of this large area it is probable that the angular convolution on the left side, in right-handed people, stores up and interprets visual word memories. If visual memories are stored in both hemispheres, they are made available from the right hemisphere only through the corpus callosum and visual word memories are stored only in the left hemisphere. (Again I am speaking of right-handed people; Figs. 105 and 142.)

The anterior part of the left frontal lobe, called by clinicians the *prefrontal area,* appears to subserve specially the purposes of volition, choice, self-control, and other higher intellectual functions; but while in some way this left prefrontal area may be the headquarters for will, intellect, and emotional control, it is dependent on the perfect coördination and interaction of the whole cerebral mechanism, more especially in the left auditory area. While tumors of the left prefrontal region are apt to be associated with change of character, failing will power, errors in intellectual and moral judgment, and lack of emotional control, failing mentality or dementia is more often the result of gradual atrophy of the whole cerebral cortex. The emphasis laid upon the relation of the left cerebral cortex to the higher sensory and motor processes in right-handed people explains the contrast between Figures 142 and 144; the so-called "silent" area in the left hemisphere is comparatively small, but in the right hemisphere it is rather large. This means that there is a much more extensive area in the right hemisphere where a lesion may give rise to no clinically localizing symptoms, or perhaps no discoverable symptoms at all, than there is in the left hemisphere, always of course in the right-handed.

Aphasia. The faculty of speech and all it implies is that which above all other qualities characterizes man's mentality as contrasted with that of lower mammalia, and the development of speech in its various phases is the most definite indication of the comparative mental advancement of different races and individuals. For this reason there is no more instructive phase in the study of cerebral activity or cerebral loss than the processes underlying speech and the lesions interfering with its integrity. The student is urged to study more comprehensive articles on the physiology of speech and the pathology of loss of speech (aphasia). (Schaefer's Physiology; Luciani's Physiology.) Here only a few outstanding points can be alluded to in the briefest terms.

The whole subject can be best understood by keeping always in mind the way in which speech is learned. First, the child stores up auditory

memories of words, associating them with states of feeling, more or less complex sensory impressions of external objects and motor results. Soon he learns to imitate these sounds, again associating the sounds he produces with a whole train of sensory and sensory-motor experiences (stored cell memories). Not till he is three or four or perhaps seven years old does he learn to read; still later he learns to write. Underlying all these are always auditory word sensations, the first form of word expression to reach his consciousness and be stored as auditory word memories in his left superior temporal gyrus. All through life most of us think by subconsciously talking to ourselves. The child pronounces often audibly every word he writes. Many people form with their lips every word they read; most read in terms of sound; few read directly by sight. Ordinarily written symbols are not imaged forth in consciousness directly but only indirectly through the subconscious activity of the auditory cortex; we inaudibly read to ourselves and so interpret what we read. In speaking, we first formulate to our own subconsciousness what we desire to say, then call up the motor speech mechanism.

Writing, last acquired, is still more complex. We probably call up subconsciously auditory memories, formulate the motor memories of words, then visualize memories of written characters, and lastly call in the higher motor memories in the second frontal cortex to set the direct motor mechanism of the arm in action for the production of written symbols we have visualized.

With Figures 142 and 143 before him the student should try to visualize the processes involved in such a commonplace activity as seeing a rose and putting it in one's buttonhole.

The rose is seen by the calcarine area, compared with previous visual memories stored in the occipital lobe and itself stored as a visual memory; thereby are called up certain olfactory, tactile, and stereognostic memories in the uncinate and parietal gyri and the whole may be interpreted as rose by the auditory center in the superior temporal gyrus which with the aid of the inferior frontal motor word memories and angular gyrus visual word memories assembles the sensory complex as a concrete concept "rose." Hence springs in the prefrontal area a desire to pluck it, calling up motor memories (kinæsthetic images) in the superior and middle frontal gyri. These acting through the giant pyramidal cells in the strictly motor area for the arm, in virtue of the sensory impressions reaching the arm through the thalamus and posterior central and parietal cortex, cause the pulling of the rose with all the complex vol-

untary and reflex coördinating mechanism involved. Finally the frontal area conceives a desire to put it in the buttonhole of the left coat lapel, a message is sent by the left superior frontal convolution by way of the corpus callosum to the right cortex commanding the left arm to adjust the coat lapel and another to the left motor area to cause the right arm to put the rose in the buttonhole. You see at once that this involves a large number of association paths besides the cross path to the right cortex. Also though there is no actual speech there is probably involved the integrity of at least two of the speech centers. It is probable that every form of voluntary activity of any complexity involves some form of unconscious speech.

Think of the paths involved in saying "Please give me that rose." The rose is seen by the calcarine area, the image stored and compared with other similar memories in the psychic visual area of the occipital cortex, associated with previous olfactory and tactile impressions in the hippocampal (uncinate) and parietal convolutions, translated into a desire for possession and a will to express the desire in the prefrontal convolutions; the immediate impulse to grab the rose is controlled perhaps in the same area, and the higher choice made of courteous asking. Therefore, a command is sent to the inferior frontal which formulates a request to the superior temporal which by further interaction releases motor impulses which audibly vocalize the request. The greater part of the left hemisphere with many centers and association fibers is involved in the simple request for a rose. Habit creates short cuts and the patient who cannot give origin to voluntary effort may be capable of cerebral reflexes. Thus the patient who cannot voluntarily ask for or even name a rose may, by a certain reflex, smell it when it is given to him; or the patient who cannot push out his tongue when told to do so may reflexly lick his lips quite naturally.

Information of this kind may be applied to the localization of cerebral cortical and subcortical lesions. Remembering the importance of association tracts in speech and in such simple mental processes as those analyzed, one realizes that *cortical lesions will cause more simple definite single* losses while *subcortical lesions,* since they interfere with the integrity of association paths, will cause more complicated dissociation in mental processes, *especially in speech* when they occur in the left hemisphere. In the right hemisphere except when primary motor or sensory areas are involved subcortical lesions that do not interfere with callosal or pyramidal fibers are almost always "silent." Cortical and subcortical lesions mean

lesions which do not involve the internal capsule or central gray matter. As soon as the thalamus, basal ganglia, and internal capsule are involved, the clinical picture changes entirely.

In the right cerebral cortex (Fig. 144) lesions irritative and destructive of the anterior and posterior central gyri have already been dealt with. Affections of the rest of the *right frontal lobe* are not productive of localizing symptoms with the exception of subcortical lesions, softening or tumor, of the posterior ends of the superior and middle frontal; these will be considered under the heading of apraxia.

Destructive lesions of the *right superior temporal* gyrus do not cause deafness because each ear is represented in both hemispheres. An irritating lesion of the right superior temporal might cause an auditory aura chiefly referred to the left ear followed by twitchings of the left side of the face and tongue, becoming afterward generalized into clonic convulsions of the whole body. The auditory aura would help to guide the examiner to the seat of the lesion and the collateral symptoms might indicate the side affected. A destructive lesion of the *right calcarine area* (area striata) would cause blindness of the right half of both retinæ (Fig. 105), or the left half of both fields of vision. As the fovea centralis has double representation, the patient may not be aware of his blindness. Irritating lesions here may cause flashes of light which occupy the left half of the field of vision and will be referred by the patient to the left eye. It is doubtful to what extent visual memories are stored by the right hemisphere in the right-handed.

The cortical center for *smell* is in the hippocampal gyrus (Figs. 141 and 145); it is mainly homolateral. Destructive lesions may be impossible to detect owing to bilateral representation. Irritative lesions may cause subjective olfactory illusions referred probably to the homolateral nostril, hallucinations of an offensive odor often accompanied by a peculiar dreamy state characterized as uncinate fits; an olfactory aura in such a case may precede a general epileptic seizure. The rest of the right hemisphere is silent for cortical and subcortical lesions, unless neighboring structures be involved such as the right optic tract, other cranial nerves or callosal fibers.

Left hemisphere. All that has been said of the immediate motor and sensory cortical areas in the right hemisphere holds for the left with corrections for the side affected. To these must be added other symptoms belonging to areas which in the right-handed receive special training (see Figs. 142 and 144). The effects of lesions of the anterior part of the posterior central convolutions and the area surrounding the horizontal limb of the posterior central fissure have been described.

Motor apraxia (Figs. 142 and 173). Lesions of the left frontal lobe produce the symptom of motor apraxia. The whole study of apraxia deals with one of the most interesting phases of cerebral activity, and the student is urged to read "A Contribution to the Study of Apraxia," by S. A. K. Wilson, *Brain,* Vol. 31. All that is said here on the subject is based on this article. A patient may have no trace of paralysis and yet have voluntary purposive movement seriously interfered with by many conditions which make difficult or impossible the formation of the purpose to act or which prevent the proper execution of the willed action. For purposes of illustration suppose that a patient is handed a cigar and a matchbox by his doctor and told to smoke. He may not be able to do so for several reasons.

1. *Cortical blindness, cortical deafness, and cortical sensory paralysis* may cut off ingoing impressions. He may be blind and not see the cigar, etc., may be deaf and not hear the command, or he may be unable to feel the box and cigar in his hand. In the last case there is a lesion of the posterior wall of the central sulcus.

2. *Agnosia.* Mind blindness, mind deafness, etc. The patient hears, sees, feels, but because of a cortical or subcortical lesion in the left occipital lobe he may not recognize that what he sees is a matchbox and cigar; or from a similar lesion in the left superior temporal gyrus he may hear the command but not be able to understand its meaning, from loss of auditory word pictures. A subcortical lesion somewhere in the neighborhood of the left insula and external capsule may interfere with association fibers or cortical combinations and so prevent him from combining his visual, auditory, and sensory impressions with previous memories so as to form the concrete idea of a matchbox and cigar and their uses.

3. *Ideational agnosia* is allied to No. 2; there is interruption between the sensory areas mentioned in 2 and the association area in the left supramarginal gyrus which may prevent a proper mental conception of all that is implied in the objects presented to the patient. This is a frequent result of general cortical deterioration such as occurs from lessened arterial supply, due to arteriosclerosis or endarteritis of the middle or anterior cerebral artery.

4. *Cerebral ataxia.* A lesion of the posterior central convolution may render the corresponding hand awkward in its movements through lack of the sensory impressions of form, position, weight, movement, which underlie all motor activity. This defect can be partly compensated for by sight. The patient may find it impossible to execute the order with

his eyes closed, but may do it more or less awkwardly with his eyes open.

5. *Cortical apraxia* (*ideational apraxia*). The area which occupies the anterior part of the left anterior central convolution (upper three-fifths), the anterior part of the paracentral lobule, and the posterior ends of the superior and middle left frontal gyri (Figs. 142 and 143) store higher motor memories or acquired habits of muscle combinations. A lesion here may prevent the patient forming a schema of the necessary combined movements to strike a match, light a cigar, and smoke it.

6. *Subcortical apraxia* (*motor apraxia*), Figure 173. The patient may have the idea of what he wants to do and of how to do it, but through interruption of association fibers to his left motor area from the area in front of it he may not be able to translate his idea into the appropriate action. His intentions are correct, but his motor combinations are defective, and opening or closing his eyes makes no difference. With a lesion such as those marked 1 to 5, in Figure 173, he might be able more or less perfectly to imitate the doctor if he is shown what to do.

As a subvariety of this type of apraxia the patient may be able to use his right hand perfectly, but because of a lesion of the callosal fibers reaching the right hemisphere from the left area of motor memories to the right motor arm center (Fig. 173), he may be unable to do what he wants to do with his left hand (hold the matchbox while drawing and striking a match, etc.), though he knows what he wants to do. In this case there may be no true left-handed paralysis, as he may reflexly scratch himself or perform other actions with his left hand which do not call for direct volition, such as using his fork in combination with the use of his knife in feeding himself.

7. *Perseveration.* Through defect in the left prefrontal area or interruption in association fibers therefrom, the patient may have difficulty in voluntarily starting an action, which, once begun, is fairly well carried out, or he may have trouble in giving up one form of activity for another when told to do so.

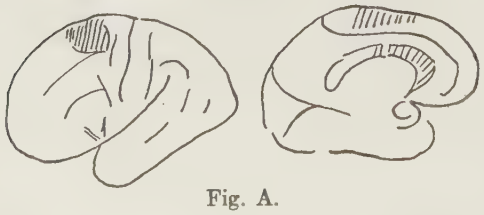


Fig. A.

CLINICAL CASES OF APRAXIA

Liepmann's and Maas's case of *Complete general apraxia of left hand;* no paralysis. Brain, vol. xxxi. Right hemisphere normal, in left hemisphere a lesion of superior frontal

convolution and of part of the paracentral lobule, and destruction of almost ⅔ of the corpus callosum on the left side.

Van Vleuten's case of apraxia. Brain, vol. xxxi, page 204. Symptoms, paraphasia, tremor right hand and leg, right tonic perseveration but right-limbed *eupraxia. Left-handed apraxia,* no paresis. Later became paralytic in right arm and leg. No optic neuritis, no other symptoms of cerebral tumor. P. M. Large sarcoma as in illustration. Tumor had destroyed white substance of right limbic gyrus and cingulum, whole of left half of corpus callosum, part of right genu of corpus callosum and white center of left frontal lobe. Cortex of third left frontal convolution intact.

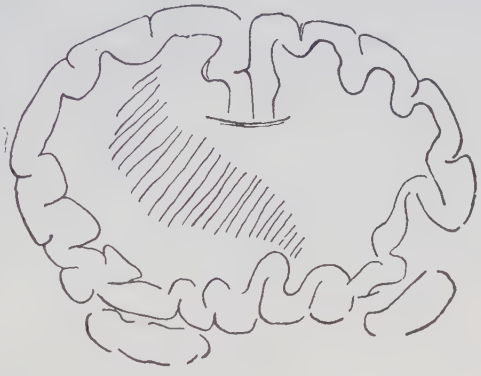


Fig. B.

Hartman's case of apraxia. Brain, vol. xxxi, page 205. Tumor of left frontal lobe involving left half of callosum, destroying genu and sending process through to white substance of right frontal lobe. Implicated lenticular and caudate nuclei and pressed upon internal capsule. Cortex of posterior ⅓ of middle and superior left frontal gyri intact. Clinically, patient lost all spontaneity, lay quiet for hours with eyes closed. All movements in response to sensory stimuli very deliberate. No weakness of right arm or leg. Complete apraxia for right hand movements, complicated movements of left hand apraxia.

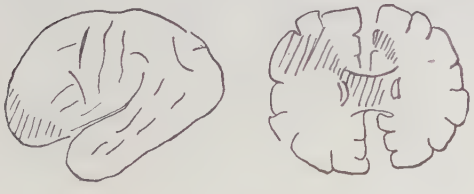


Fig. C.

Hartman's 2nd case. Tumor of corpus callosum spreading along mesial aspect of left hemisphere as far as calcarine fissure. Central paths and projection fibers not involved. Complete apraxia of left hand. Movements of right hand slow, requiring the assistance of the eyes. Neither arm paretic. Left hand not aided by eyes.

Fig. D.

Case 3. Cyst in middle of superior frontal lobe injuring cortex of middle frontal and reaching cortex of inferior frontal. Patient unable to grimace or imitate grimaces. Diffi-

culty in making given movements with tongue. Right arm normal; left frequently failed to perform set movements. In handling objects no defect on right side; on the left amorphous or akinetic (ineffectual) movements were made. Bilateral simultaneous use of hands perfect. Beckoning and threatening movements good on both sides.

Fig. E.

Aphasia. (Fig. 142.) In its various forms aphasia is a special variety of apraxia. All forms of speech hinge on the cortical integrity of the auditory word memory center in the left superior temporal convolution, or the subcortical connections by association fibers between this and visual and motor centers.

Owing to the bilateral representation of each auditory (cochlear) nerve, a lesion of the *left superior temporal convolution* does not cause deafness, but it may cause total or partial inability to interpret auditory word impressions. The patient may hear words but not know their meaning; this has very far-reaching effects. A patient who is deaf from ear or nerve defect suffers little mental deterioration, for he can still read, still commune with himself, still speak (subconsciously) to his own word interpreting center. In the deaf mute who has learned to talk with hands or lips another center is trained. Any extensive lesion of the left superior temporal convolution interrupts this process of subconscious self-communion and serious intellectual deterioration may result.

A lesion of the left internal capsule or *lower end of the left anterior central convolution* may cause difficulty in articulation (dysarthria) which, however, may be largely corrected by training of the opposite motor area.

Destruction of the *posterior end of the left inferior frontal gyrus* may prevent the conception of word movements and so result in loss of the power of speaking, without actual paralysis of the tongue, lips, or larynx. This is known as motor aphasia.

A center for writing has long been believed to exist in the *posterior end of the second left frontal convolution,* and late discoveries in the mechanism of motor apraxia seems to support this view. So closely, however, are speech and auditory word memories associated with the power to write that this power is often lost when a subcortical lesion interferes with speech, without any apparent affection of the supposed center for writing. Some uncertainty still surrounds the supposed center in the *angular convolution* for visual word memories (reading), and it is still doubtful if a purely cortical lesion destroying the left angular convolution

would produce alexia, or inability to interpret written characters, apart from the interruption of deeper association fibers between this convolution and the area striata on the one hand and the left superior temporal on the other. Hence when a surgeon operates for a tumor which causes aphasia he makes a wide exposure of the brain, including the superior temporal and possibly the posterior end of the inferior frontal and angular convolutions (one or both) as well.

HENRY HEAD'S CLASSIFICATION OF APHASIAS

It seems inadvisable to close even such casual allusion to the cortical speech mechanism without an attempt to give at least some faint indication of Head's great work on war aphasias.* (Fig. 226.)

The psychic processes involved in speech are called by Head "symbolic formulation and expression." This seems sufficiently comprehensive to include all the means used to formulate our ideas and communicate them to each other.

The cortical area whose integrity is necessary for the normal performance of symbolic formulation and expression is included in the posterior end of the inferior frontal gyrus, the lower two-thirds of the anterior central gyrus, the supramarginal, angular and superior and middle temporal convolutions together with the projection fibers entering and leaving these and the association fibers joining them.

While certain districts of this area are nodes providing for the integration and rearrangement of afferent and efferent impulses involved in the reception, formulation and emission of language symbols (so-called speech centers) no one part of this area acts alone. Normal symbolic formulation and expression depends on the coördinated activity of the whole mechanism and the behavior of a patient with any localized defect of the speech area of the cortex is the result of his effort to make use of his defective mechanism.

When certain areas of integration (so-called centers) are injured and the rest escape, there is always an initial period of diaschisis when the whole speech mechanism is so crippled that no types can be differentiated; but after this initial period of diaschisis has passed off, the remaining defects of language tend to be grouped within certain types in which certain defects of speech more or less dominate. These dominant defects have been indicated in Head's use of the terms *verbal aphasia,* when the

* *Aphasia and Kindred Disorders of Speech,* Henry Head. The Macmillan Co., 1926.

posterior end of the inferior frontal convolution is injured, *nominal aphasia,* when the angular gyrus has suffered, *syntactic aphasia,* when the superior or middle temporal is involved, and *semantic aphasia* when the supramarginal gyrus is at fault.

The chief characteristics of each of these types of aphasia are as follows:

1. Chief characteristics of *verbal aphasia* (Frontal type). There is at first profound loss of speech. The patient is dumb or his vocal speech is limited to single words so constantly used as to have become automatic, *e.g.,* "yes," "no." He is also capable of emotional ejaculations, *e.g.,* "Oh dear!" "Damn!" As the patient recovers words are badly pronounced; but their nominal values are appreciated. There is no tendency to substitute words of wrong meaning.

The vocabulary is limited. The patient speaks slowly with effort. Long words may be slurred. The difficulties are not purely difficulties of articulation (dysarthric). To the last there is verbal hesitation and syncopated rhythm (tendency to omit connecting words or syllables).

2. Chief characteristics of *nominal aphasia* (angular gyrus type).

There is inability to find verbal symbols of appropriate significance.

During the first few weeks the patient may be almost dumb, but the few words he has are comprehensible, and if he utters a phrase the syntax is not defective.

Later, the essential difficulty is a want of power to use names correctly and to select words of appropriate meaning.

The patient uses descriptive terms for names, *e.g.,* the color black was described by a patient as "what we do for the dead," and Head had a female patient who, unable to find his name, called him "hairpin," "top knot." Such patients give long descriptions of their difficulties.

3. Chief characteristics of *syntatical aphasia* (temporal type). The tendency to talk jargon makes the distinction easy. Speech is voluble and very rapid. The words are strung together without articles, conjunctions or prepositions, so that there is want of grammatical coherence. Phrases and sentences repeated after the examining physician tend toward the same defects.

Naming objects may be unintelligible, but names are not lost as the patient picks out the right names on printed cards.

Articulated speech is at first incomprehensible, but the words soon become intelligible though the phrase is faulty.

4. Chief characteristics of *semantic aphasia* (supramarginal type).

There is little disorder in articulated speech. The patient has no difficulty in finding names or forming words, and he can repeat what is said to him, but when he attempts to repeat a story essential elements in the series of happenings are left out, for he cannot retain in his mind the total conception. Such patients talk rapidly as if afraid of forgetting what they want to say. If they stop to think they become confused.

With very few exceptions the left hemisphere is involved in right-handed aphasics and vice versa.

In each type neighborhood symptoms may be of value in localization. Thus all but the most superficial affections of the angular gyrus are apt to be associated with injury to the underlying optic radiation, and consequent right homonymous hemianopsia. In some temporal lesions also there may be injury to the optic radiation, and deeper temporal lesions are apt to involve the uncinate gyrus with resulting subjective sensations of a bad smell together with chewing movements and a peculiar dreamy state characteristic of uncinate fits (the uncinate type of Jacksonian epilepsy).

So too inferior frontal lesions may be associated with right-sided facial spasm or involuntary movements of the fingers, or facial paralysis of the upper motor neuron type if the lower third of the anterior central gyrus be involved in the lesion, and in this case verbal defects will be associated with difficulties in articulation (dysarthria), due to paralysis of the lips, tongue, larynx, etc.

Lesions of the supramarginal gyrus may be associated with attacks of numbness and tingling in the face and arm associated with damage to the neighboring posterior central convolution.

Fresh injuries and progressive lesions always give rise to combinations of all forms of language defects and it is only when the lesion has been long stationary and all evidence of diaschisis has passed away that the particular type can be recognized and any hope of localization of the lesion entertained.

The occurrence of Jacksonian fits and any depression of the general health or lowering of the psychic tone of the patient by worry is likely to be accompanied by temporary increase of symptoms without necessary extension of the lesion. Such relapses are readily recovered from when the physical and psychic well-being of the patient has been restored.

Vascular lesions in old people seldom admit of accurate localization, as all forms of speech are apt to be seriously handicapped and such patients, owing to their age and consequent circulatory and general dis-

abilities, have little power to make the best of the crippled speech mechanism and become very impatient and uncoöperative under examination.

No superficial examination of an aphasic patient will suffice to differentiate the type of aphasia from which he is suffering. Only careful and repeated use of graded tests such as Head employs, used over long periods of time, can yield accurate diagnostic data. It is to be remembered that results vary greatly from day to day with the psychic and physical condition of the patient. Much depends on perfect quiet in the surroundings and willing coöperation on the part of the patient. Fatigue must be avoided.

The results of work on war aphasias recorded by Marie and Foix (*Revue Neurologigue,* 1917) are very similar to Head's in localization and type, but their nomenclature is by anatomical regions and their interpretation of recorded symptoms somewhat different. Similar work has been done in Germany by Gelb and Goldstein.

DECEREBRATE RIGIDITY IN MAN

Under this heading in *Brain,* 1920, S. A. K. Wilson calls attention to a group of cases in which from various causes the parts above the tentorium had been rendered inactive, the parts of the brain and cord below the tentorium still functionating but without higher control.

Cause. In all the cases in the first group, the symptoms of decerebration came on acutely. There was in one tuberculous meningitis at the base with moderate hydrocephalus, in one an abscess of the temporosphenoidal lobe which ruptured into the temporal horn of the lateral ventricle with infection of both lateral ventricles and the third ventricle, and in the others hemorrhage into the lateral and third ventricles; in one of these the hemorrhage into the third ventricle was due to a mesencephalic glioma.

Symptoms. With a previous rather indefinite history of headache, vomiting, and choked disc, the acute symptoms were ushered in by somnolence passing into coma, and by general rigidity of the type recognized in experimental animals in which the mesencephalon has been divided, namely, rigidity of all the limbs in extension. The legs were extended from hip to ankle, the toes flexed, the feet in plantar flexion and soles turned inward. The arms were adducted at the shoulder, strongly extended at the elbow, the forearms were hyperpronated till the dorsal surfaces of the hands looked inward, the fingers flexed and thumb adducted. This attitude was exaggerated from time to time by tonic

convulsions, in which the extension and pronation were accentuated. Usually the fits were very frequent and could be induced by any disturbance.

Respiration was usually slow, six to eight per minute, the inspiration quick and the expiration long drawn out. The respiratory difficulty was much exaggerated during the fits.

Patients invariably died in from six to twenty-eight days after the beginning of the rigidity. Two cases are recorded by other observers of similar general rigidity with tonic convulsions in infants who after death showed marked forebrain aplasia, the thalamus and basal ganglia being apparently normal (Sutherland and Paterson, *Quarterly Journal of Medicine,* Vol. 7).

With these cases in mind it may be well to review present knowledge regarding decerebrate rigidity in animals. It occurs after section of the brain stem just above the superior colliculi or through the posterior end of the thalamus and continues during successive sections till section is made at the pontobulbar junction. It is regarded by Sherrington, Wilson, *et. al.,* as a release phenomenon depending on the activity of centers lying between the two levels of section, which are released from the control of centers higher up. Stimulating and destructive experiments together seem to indicate that the activating centers are the red nucleus, acting mainly heterolaterally probably by way of the rubrospinal tract, and Deiters' nucleus and perhaps other vestibular nuclei, acting mainly homolaterally through the vestibulospinal tract.

With the exception of a small area of the cortex of the superior vermis, stimulation of which inhibits rigidity, the cortex cerebelli has an adjuvating influence over the red and vestibular nuclei, but rigidity is not abolished by destruction of the cerebellar hemispheres, nor by section of both vestibular nerves nor destruction of both inner ears. It is inhibited homolaterally by section of the ventral spinocerebellar tract but not by section of the dorsal spinocerebellar tract or of the restiform body. It is inhibited homolaterally by section of the brachium conjunctivum, perhaps due to the section here of the ventral spinocerebellar tract. It is inhibited by stimulation of the fronto-pontine fibers in the internal capsule. Section of the posterior nerve roots to a limb inhibits rigidity in that limb. Bilateral degeneration of the globus pallidus causes general rigidity, and unilateral degeneration of the globus pallidus causes heterolateral rigidity.

It would appear then that muscular rigidity is dependent on the integrity of the spinal reflex arc and of the ventral spinocerebellar path which

sends collaterals to Deiters' nucleus and ends in the superior vermis. It is caused by the uncontrolled activity of the red nucleus, Deiters' nucleus, and perhaps other nuclei in the brain stem between these two levels. On the whole muscular rigidity receives an adjuvating influence from the cerebellum, though there is a small inhibiting center in the superior vermis. The globus pallidus is inhibiting; its influence is exerted either on the red nucleus or directly on the spinal reflex arc through a lenticulo-rubro-spinal or other descending path (lenticulo-nigro-spinal?). There also appears to be an inhibiting path from the frontal cortex by way of the fronto-pontine tract, the opposite brachium pontis and the cortex of the superior vermis.

These facts would seem to underlie the correct interpretation of partial rigidities such as occur in certain cases of congenital diplegia, lenticular degeneration, and paralysis agitans. In progressive degeneration of the globus pallidus on both sides the subcerebral centers are released from pallidal control and rigidity results. It is probable that in certain cases of diplegia in children where the rigidity outstrips the loss of voluntary control and athetoid movements are present, the cause is to be looked for in the basal ganglia rather than in the cerebral cortex. In adults paralysis agitans is being more and more ascribed to disease of the globus pallidus or its relay stations, the red nuclei and substantia nigra.

SUPPLEMENT

Here follows an attempt to classify the commoner nervous diseases on an anatomical basis. It may be used by the student as a means of rapid review of the whole subject and as an aid to diagnosis.

CLASSIFICATION

Diseases of the Motor Neurons

Paralysis of motion may be due to:

A. Muscular lesions.
B. Lesion of lower motor neuron.
C. Lesion of upper motor neuron.
D. Combined upper and lower motor neuron lesion.
E. Cord disease affecting both motor and sensory tracts.
F. Interruption of the sensory side of the reflex arc.
G. Reflex causes.
H. Paralysis without known lesions.
I. Hysterical paralysis.

A. Paralysis due to muscular affections without discoverable nervous lesion; no reaction of degeneration; no fibrillar twitchings.

1. Congenital. No family tendency: amyotonia (myatonia) congenita (classification doubtful).

2. Progressive muscular weakness; familial tendency; begins early in life; pseudohypertrophic paralysis; other muscular dystrophies.

3. Myasthenia gravis; appears first in adults.

B. Paralysis due to lesions of the lower motor neuron (Fig. 99). Symptoms common to all types:

Flaccid paralysis of all muscles supplied.
Loss of reflexes for these muscles.
Reaction of degeneration (R.D.).
Muscular wasting.
Histological degeneration of paralyzed muscles.

The symptoms are always homolateral, and the distribution segmental or according to the anatomical distribution of peripheral nerves.

Unilateral lower motor neuron paralysis.

1. *Peripheral Nerve* (Fig. 99). Muscular distribution of paralysis according to the distribution of the peripheral nerve involved. Accompanying anæsthesia if lesion be complete, or paræsthesia, hyperæsthesia, hyperalgesia if lesion be incomplete, for peripheral distribution of the sensory branches of the nerve.

Trophic disturbances in skin if nerve contains cutaneous branches.

EXAMPLES

(A) *Neuritis.*

(*a*) Single nerve—sciatic neuritis, facial neuritis.

(*b*) Multiple neuritis—tendency to distribution symmetrically and at ends of extremities.

Due to exogenous poisons—alcohol (Fig. 165), lead, arsenic.

Autointoxication—diabetes, nephritis.

Infections—diphtheria, scarlet fever, typhoid, leprosy (Fig. 192), beri-beri.

(B) *Tumor of nerve trunk.*

(C) *Traumatism.*

(*a*) Pressure palsies.

Sleeping in awkward positions (Fig. 151).
Pressure from faulty position in anæsthetic administration.
Crutch palsy of axillary or radial.
Fracture palsy of radial, or axillary.
Pressure from badly-fitting splint.

(*b*) Wound of nerve.

2. *Trunk of Spinal Nerve* (Fig. 99). (In cranial nerve it may be the intramedullary nerve roots.)

Characterized by segmental distribution of the paralysis and of the sensory symptoms.

EXAMPLES

(*a*) Traumatism of spinal nerve as by stab close to intervertebral foramen.

(*b*) Pressure on nerve trunk as by cervical rib. (D. 1 or C. 8 and D. 1.) Fig. 210, a, b, c.)

[NOTE: Traumatism to a primary trunk of the brachial plexus will produce segmental symptoms; the upper trunk represents the fifth and sixth cervical segments, the middle trunk the seventh segment, and the lower trunk the eighth cervical and first thoracic segments.]

(*c*) Pressure on a nerve trunk in tuberculous disease of the spinal column; or in tumor, fracture, or dislocation of the vertebral column.

(*d*) Birth palsy (Fig. 166).

(*e*) Nerve trunks may be involved in syphilitic or tuberculous meningitis (Fig. 191).

[NOTE: If there be pressure on or irritation of a spinal ganglion, there may be severe neuralgia or herpes for the cutaneous distribution of the sensory root.]

(*f*) In the case of a *cranial nerve,* the intramedullary nerve fila (Figs. 174 and 175) may be involved in a lesion of the brain stem.

3. *Anterior nerve root.* The distribution is segmental; there are no sensory symptoms in a pure anterior root lesion. Example: purely motor cranial nerve. The anterior root may alone be involved in a wound of the cauda equina.

[NOTE: In the syphilitic radiculitis of Degerine (Fig. 208) both anterior and posterior nerve roots are usually involved together, with consequent segmental motor and sensory symptoms.]

4. *Lesions of Nerve Cells of Lower Motor Neuron* (Figs. 153 to 158). Rapid flaccid paralysis, wasting, fibrillar twitchings (fibrillar twitchings are seldom found in peripheral lower motor neuron lesions, as neuritis), R. D., no sensory symptoms as a rule, distribution segmental, usually over several segments.

Note. Fibrillar twitchings are characteristic of slow destruction of lower motor nervous cells.

Poliomyelitis.

Acute (Figs. 153 to 159).

Chronic (including the spinal forms of progressive muscular atrophy, Figs. 160 and 162).

Polioencephalitis. Motor nuclei of cranial nerve or nerves.

Acute (Fig. 156).

Chronic. *e.g.,* bulbar palsy.

Bilateral Lower Motor Neuron Paralysis.

Most cases of paralysis of both sides of the body are due to upper motor neuron affections. There are, however, a few lower motor neuron affections of bilateral distribution.

5. *Lower Motor Neuron Paraplegia.* Flaccid paralysis, R.D., wasting, loss of all reflexes.

(a) *Peripheral polyneuritis,* especially alcoholic, tends to be symmetrical in the legs. (There is accompanying pain and anæsthesia from the sensory lesion—Fig. 165.)

(*b*) *Nerve root type.* Lesion of cauda equina by tumor or fracture. (See description of cauda equina lesions, Fig. 207.)

(*c*) *Lesion of nerve cells of anterior horn.* Acute poliomyelitis may assume a paraplegic type affecting both lower extremities. In poliomyelitis of the sacral segments the sphincters will be involved if the third and fourth sacral segments are implicated.

(*d*) *Landry's paralysis.* This is an acute paralysis usually beginning in legs and ascending in two or three days or as many weeks to the cranial nerves, more rarely beginning above and descending, frequently causing death in three or four days to a month. It is due perhaps to different neurotoxins, and may be a rapidly advancing poliomyelitis of general distribution or an equally widely distributed peripheral polyneuritis. Paralysis, flaccid. Usually no R. D. as there is seldom time for it to develop. Sensory symptoms usually absent; sometimes pain is present.

(*e*). *Syringomyelia,* especially in the cervical region, may affect the LMN cells of both anterior gray columns, producing a brachial LMN diplegia (paralysis of both arms). (Figs. 215 and 216.)

[Note: In vascular and other lesions of the brain stem, syringomyelia (Fig. 187), hematomyelia (Fig. 213), myelitis, embolism and thrombosis of the cord, cord tumors (Figs. 184 and 185), and in amyotrophic lateral sclerosis (Fig. 186), lower motor neuron lesions may be found mixed with lesions of sensory nerve roots or tract lesions of the brain stem or cord. See combined types.]

C. Upper motor neuron paralysis.

All upper motor neuron paralyses have these features in common:

More or less loss of voluntary motion for the parts involved, according to the extent and completeness of the destruction of the pyramidal path.

Loss of skin reflexes for the part involved, notably loss of cremasteric reflex in leg, and abdominal and epigastric reflexes in trunk. (This is an inconstant sign and is most valuable if confined to the hemiplegic side in one-sided paralysis.)

Extensor plantar response (Babinski's sign) if the leg be involved.

Increase of tendon reflexes for part or parts involved (jaw jerk, triceps jerk in arm; knee jerk, tendo-calcaneous jerk, ankle clonus in leg).

Peripheral muscles and skilled movements are most affected. Spinal and respiratory muscles, also the muscles of tongue, jaw, upper face, larynx, have a bilateral pyramidal control and are only weakened, not paralyzed.

More or less rigidity of the paralyzed limb. See "general considerations."

Usually no wasting of muscles occurs till later from disuse.

Never R.D. in pure upper motor neuron lesion.

Upper Motor Neuron Unilateral Paralysis.

1. *Cortical Lesions—Lesions of the Anterior Central Convolution.* Arteriosclerosis with thrombosis, embolism, or hemorrhage of anterior cerebral artery (Fig. 146) (leg area), or middle cerebral (Fig. 146) artery (face and arm area); tumors, traumatism (Fig. 167).

Monoplegic type (paralysis on side opposite to the lesion). Tendency to irritative symptoms of neighboring cortex with convulsive seizures and paræsthesia, sensory hallucinations.

Rigidity absent or slight. Contractures appear late or not at all. More or less loss of sense of position.

2. *Subcortical Lesions*—lesions of the white matter under the anterior central convolution and above the basal ganglia. This is an ischæmic area where the cortical and basal arteries both terminate (Fig. 148). For this reason perhaps the usual lesions are ischæmic softening, or abcess. Tumors also may occur here.

The lesion is apt to be more nearly a complete interruption of pyramidal fibers, hence the paralysis is more of a hemiplegic type; convulsions are exceptional. If the lesion be in the left side of the brain, there is apt to be a permanent aphasia owing to interruption of association fibers concerned in speech production. (See aphasia.)

Subcortical motor lesions may be accompanied by anæsthesia of the cortical type (tactile and muscular judgments). Such cases show a tendency to greater rigidity than cortical lesions.

3. *Capsular Lesions* (Fig. 168, a). Usually a hemorrhage due to rupture of a basal artery (lenticulo-striate or lenticulo-thalamic in 60% of cases). Sometimes a softening owing to arteriosclerosis, thrombosis, or embolism of a basal artery. The usual type of cerebral apoplexy or "paralytic stroke."

Type hemiplegic, without convulsions, the upper face escaping. The eyes are involved only temporarily. The upper face is always weakened and may be paralyzed for special movements such as winking with the eyelid of the paralyzed side (Fig. 193). The masticatory and tongue muscles are weak but not completely paralyzed owing to bilateral supply. In left-sided hemorrhage in *right-handed* people there is motor aphasia (anarthria) which tends to improve. The lesion usually destroys more or less of the pyramidal tract in the internal capsule, and also the lenticular nucleus or lenticular efferent fibers; therefore the rigidity is marked (see general considerations).

Rigidity of the leg in extension (Fig. 196). Rigidity of the arm in adduction and medial rotation of the arm, semiflexion and semipronation of the forearm, flexion of the fingers (Figs. 194 and 195). Increase of tendon reflexes, loss of cutaneous reflexes, extensor plantar response.

Usually there is more or less loss of sense of position, localization, and tactile judgment owing to interruption of thalamo-cortical fibers.

If the thalamus be involved there may be in addition to the paralysis:

(*a*) Marked sensory loss.

(*b*) Posthemiplegic choreoathetoid movements if the pyramidal lesion be limited.

(*c*) Hemianopsia if pulvinar or optic radiation be involved.

(*d*) Thalamic pain(?).

4. *Lesions of the Mesencephalon* (Fig. 180). Hemorrhage, softening due to thrombosis or embolism, tubercle, gumma, or other tumor within the mesencephalon or pressing upon it. The type is hemiplegic, being an upper motor neuron paralysis for the opposite side of the body.

(a) *Lesion of the basis pedunculi* (Fig. 180). This is a pure pyramidal lesion (upper motor neuron paralysis) for the opposite side of the body; the upper face, masticatory muscles and tongue are only paretic as they have a bilateral pyramidal supply. The corresponding lenticulo-rubrospinal tract which lies in the opposite tegmentum escapes; therefore the paralysis of the opposite limbs should be accompanied by little rigidity (see general considerations).

In the mesencephalon pyramidal fibers for the cranial nerves separate off as aberrant pyramidal fibers, and therefore occasionally cranial nerves escape.

The emerging fibers of the third nerve can scarcely escape, so there will be lower motor neuron paralysis of the third nerve on the side of the lesion (Fig. 180).

There will be no sensory disturbance (contrast Figs. 180 and 181).

(*b*) *Mixed lesion of basis pedunculi and tegmentum* (combine Figs. 180 and 181). This produces upper motor neuron paralysis of the opposite side of the body with additional symptoms varying with involvement of the following tegmental tracts and nuclei.

Red nucleus. The paralysis is accompanied by rigidity from loss of the lenticulo-rubrospinal control of muscle tone. Cerebellar symptoms are prevented or masked by the pyramidal lesion. Cerebellar tremors are characteristic of a red nucleus lesion but are incompatible with a marked pyramidal lesion to the same side.

Lemniscus medialis. There is contralateral sensory loss, all forms, on the same side as the paralysis.

Third nerve fila or nucleus. Homolateral ptosis, dilated pupil, external strabismus.

Brachium conjunctivum. Homolateral tremors, asthænia, atonia, asynergia, ataxia (of cerebellar type), choreoathetoid movements.

Rubrospinal tract. This crosses above this level and will give homolateral symptoms if involved here; hence there are homolateral cerebellar symptoms. It seems probable that the cerebellar atonia would counteract the rigidity which should follow the loss of the lenticulo-rubrospinal control.

[Note: An intramedullary tumor blocking the aqueductus cerebri may give all the symptoms mentioned with the addition of general convulsions owing to increased intraventricular pressure from obstructed drainage from the cerebral ventricles.]

5. *Pyramidal Lesion in Upper Pons* (Figs. 182 and 169, a). (The same lesions are possible as those mentioned under Mesencephalon—C. 4.) There is hemiplegia of the opposite side of the body, the eyes escape, the upper face, masticatory muscles and tongue are weakened.

(*a*) *Pure basilar lesion* (Figs. 182 and 183). There is rigidity as in C. 4.

Theoretically as the cortico-ponto-cerebellar fibers for both sides may suffer severely there should be symptoms corresponding to a lesion of the brachium pontis. These are not described in reported cases and may be absent or masked by the hemiplegia. Also brachium pontis symptoms are very obscure clinically.

(*b*) *Mixed basilar and tegmental lesion of the upper pons* (Fig. 178). Besides the hemiplegia the following additional symptoms may be found according to the structure involved.

Fifth nerve nucleus or emerging roots. Homolateral lower motor neuron paralysis of the fifth nerve with paralysis of the muscles of mastication and fifth nerve anæsthesia.

Lemniscus medialis (Fig. 178). More or less hemianæsthesia on the opposite side of body.

Medial longitudinal bundle. Opposite eye cannot be turned to the side of the lesion in conjugate deviation (Fig. 182).

6. *Lower Pontine Lesion* (Figs. 178 and 183). The causes are similar to those already mentioned. In addition to the pyramidal tract the emerging fila of the sixth nerve, or of the sixth and seventh nerves are usually involved.

There may be typical "crossed paralysis," *i.e.*, an upper motor neuron paralysis of the opposite side of the body not involving the face, as the pyramidal fibers to the side of the face opposite to the lesion have crossed higher; this paralysis is accompanied by a lower motor neuron lesion of the sixth or the sixth and seventh nerves on the same side as the lesion (Fig. 178).

The prominent symptom is hemiplegia of the opposite side of the body. If the lesion extend upward above the decussation of the pyramidal fibers to the face the hemiplegia may include the face. The character of the rigidity is the same as that mentioned for the mesencephalic lesions. In addition there may be:

(*a*) If sixth nerve roots are involved, there is lower motor neuron paralysis of the abducens oculi with internal strabismus of the homolateral eye, *i.e.*, that on the side of the lesion (Fig. 178).

(*b*) If seventh nerve roots or nucleus are involved, there is lower motor neuron paralysis of the face on the side of the lesion. The whole face is involved. There is inability to close the eyelids, the brow is smooth, the angle of the mouth droops, the mouth is drawn to the opposite side, the platysma is paralyzed, there is R. D. and wasting (Fig. 178).

(*c*) If the medial lemniscus is involved, there is loss of muscle, compass, and tuning-fork sense for the opposite side of the body (Fig. 178, b).

(*d*) If the medial longitudinal bundle is involved, there is weakness of the opposite eye for conjugate deviation to the side of the lesion (Fig. 178, a and b).

(*e*) If the vestibulospinal tract is involved, there is marked atonia on the side of the lesion (Fig. 178, b).

(*f*) If the spinothalamic tract is involved, there is loss of pain, heat, and cold for the opposite side of the body, which may include the face if the trigeminothalamic tract is included (Fig. 178, b).

(*g*) If the spinal tract of the fifth nerve is involved, there is facial anæsthesia on the side of the lesion (Fig. 179).

(*h*) If the lesion spreads high enough to interrupt the pyramidal fibers to the

opposite seventh nucleus, the hemiplegia of the opposite side of the body may include an upper motor neuron facial paralysis.

7. *Limited Lesion of the Pyramid in the Medulla Oblongata* (Figs. 174 to 177).

Exceptionally thrombosis of the anterior spinal artery at its origin may result in a lesion confined to the pyramid of one side of the medulla oblongata (Fig. 174).

A lesion limited to the pyramid causes upper motor neuron paralysis of the opposite side of the body, the head escaping. Spasticity as described under mesencephalic lesion (C. 4).

Other symptoms will vary in accordance with the involvement of the following structures in wider lesions (Figs. 174 to 177).

(*a*) The emerging fila of the hypoglossal nerve are usually involved in such lesions (Figs. 174 and 175). There will be lower motor neuron paralysis of the half of the tongue on the side of the lesion with rapid wasting. The paralysis is complete, not incomplete as in upper motor neuron paralysis of the tongue.

(*b*) The medial lemniscus may be involved with resulting loss of muscle sense, tuning-fork and compass sense, on the opposite side of the body (the same side as the hemiplegia).

(*c*) If, as in Figure 175, the spinothalamic tract is involved, there will be loss of pain, heat and cold for the opposite side of the body.

In case of Figure 175 the spinal tract of the fifth nerve is so near the spinothalamic tract that it is likely also to be involved. If so, there will also be probable facial anæsthesia for all forms of sensation, especially heat and cold on the side of the lesion.

(*d*) If the emerging fila of the accessory nerve be involved, there will be paralysis of the homolateral vocal cords.

(*e*) If the restiform body or spinocerebellar tracts be destroyed, there may be homolateral cerebellar symptoms, as atonia, asthænia, asynergia, tremors or choreo-athetoid movements on the side of the lesion. This will be still better marked if the vestibulospinal tract be interrupted. For the position of these tracts see Figure F of the long diagram.)

(*f*) In the medulla oblongata there is a center controlling the pupil dilator center in the eighth cervical and first thoracic segments of the cord (compare Fig. 94). If this be involved there will be homolateral contraction of the pupil and drooping of the eyelid (paralysis of the palpebral muscle of Müller).

[Note: Any gross lesion of the medulla oblongata is incompatible with life on account of paralysis of the respiratory center.]

8. *A Half Lesion of the Cord* causes a homolateral, one-sided paralysis below the lesion, as the motor decussation has taken place in the medulla oblongata (Figs. 109 and 110).

Causes. One-sided tumor, one-sided traumatism as from stab wound or gunshot concussion (half lesions were produced in the late war by shrapnel). Certain types of syringomyelia.

Results. A more or less typical Brown-Sequard paralysis (Figs. 109, 110 and 214).

Symptoms. Homolateral upper motor neuron paralysis of the body below the lesion. The paralysis is spastic, either with the spasticity characteristic of any incomplete cord lesion, or due to that, plus the influence of the vestibulospinal tract, which may escape owing to its isolated position.

There is a homolateral lower motor neuron paralysis of the segmental type due to destruction of the anterior gray column cells at the site of the lesion.

There is homolateral lower sensory neuron anæsthesia of segmental type due to destruction of the sensory nerve roots at the site of the lesion. There is homolateral loss of muscle sense, tuning-fork sense, and compass sense owing to destruction of the posterior column (sometimes tactile sense is lost homolaterally), for the body below the leion. There is a heterolateral loss of pain, heat and cold and perhaps of touch in the body below the lesion.

For other particulars see the discussion of Brown-Sequard paralysis.

9. *Upper Motor Neuron Bilateral Paralysis.*

(*a*) Complete congenital absence of the pyramidal tracts or whole cerebral cortex or the corona radiata as in some cases of congenital hydrocephalus.

(*b*) Defective development of the pyramidal tracts (Little's disease, Fig. 200). This occurs when premature infants are paralyzed on both sides and spastic from birth. It is not the only cause of Little's syndrome, *e.g.*, there may be a double monoplegia affecting both legs from bilateral cortical hemorrhage occurring during labor.

(*c*) As the causes that predispose to cerebral hemorrhage and softening (high blood pressure, arteriosclerosis, syphilitic endarteritis) are general diseases, it is not uncommon to have a paralytic stroke affecting one side, soon followed by a second stroke affecting the opposite side. Thus we may have:

(1) Double monoplegic diplegia from a double cortical lesion.

(2) Two capsular lesions affecting first one, then the other internal capsule, giving rise to a double hemiplegia.

10. *Paraplegia* (Figs. 170, 202 to 207). This is due to interruption of the upper motor neuron (lateral cerebrospinal tract) on both sides of the spinal cord.

Symptoms common to all forms. Under ordinary conditions in civil practice a complete transverse lesion of the cord in man is followed by a flaccid paralysis and complete anæsthesia for all parts below the lesion. Muscles waste and lose faradic excitability. Sphincters lose their tone; the only sign of cord action is occasional slight skin reflexes. No degeneration occurs in the ventral horn cells of peripheral nerves. See general considerations.

Incomplete lesions. After a period of flaccidity acute cases exhibit paralysis in proportion to the interruption of the cerebrospinal tract, and spasticity in proportion as the vestibulospinal tract is spared and the pyramidal and rubrospinal tracts are destroyed. The tone of the cord below the lesion will also largely depend on the success in preventing bed sores and bladder infection. (See spasticity in the introduction.) The earliest sign of incompleteness of the lesion in acute cases or of returning spinal reflexes is a returning Babinski's sign.

In *chronic transverse cord lesions* (Fig. 170 and history) the earliest sign of interruption of the cerebrospinal tract is a sense of weight on motor effort, increasing paresis and Babinski's sign. Later the patellar and other deep reflexes are

increased, and there is increasing rigidity. If there be preëxisting tabes dorsalis or diabetes, these reflex signs may be permanently absent. Also in combined sclerosis (Fig. 186) and in amyotrophic lateral sclerosis (Fig. 171) reflexes and rigidity may be more or less absent.

(*a*) *Flaccid paraplegia.* This is the usual form of paraplegia in complete transverse lesions of the cord during the period of shock. Flaccid paraplegia may be due to:

(1) *Myelomalacia,* or softening of the cord, from thrombosis or embolism of the spinal vessels.

(2) *Transverse myelitis,* usually of an infective character. (The term myelitis is confined by the writer to inflammatory affections.)

(3) *Tumors* of the cord and meninges or of the vertebræ or tuberculosis of the vertebræ when the lesion is complete. There is usually an earlier period of spasticity. Pain of radicular distribution is common.

(4) *Traumatism.*

(*a*) Gunshot injuries.

(*b*) Fractures of spine.

In all these forms of flaccid paraplegia the flaccidity is in proportion to the completeness and the acuteness of the lesion. There is loss of all motion, all sensation, all reflexes below the lesion. There is no R. D., no muscular degeneration. There is atrophy from lack of use. There is lower motor neuron paralysis of the segmental type at the level of the lesion.

(*b*) *Paraplegia of the spastic type* (Figs. 202 to 207).

(1) *Acute invasion.* Incomplete transverse lesion. As a rule the paralysis is flaccid at first and only becomes spastic after the spinal shock has passed off. It may be due to myelomalacia, hematomyelia, or traumatism.

(2) *Less acute invasion.* The paralysis is of the spastic type from the first, paresis passing into paralysis. It may be caused by:

Circumscribed syphilitic thickening of the pia mater.
Tuberculosis of the spine or more rarely of the cord.
Tumors of the cord, meninges or vertebræ (Figs. 184 and 185).

(c) *Slowly progressive lesions.*

Symptoms. Those of a slowly progressive, often intermittent paresis with a positive Babinski passing into paralysis with rigidity; sometimes the rigidity is in excess of the paralysis. No lower motor neuron lesion accompanies this type.

(1) Purely primary sclerosis of the lateral cerebrospinal tracts called *primary lateral sclerosis* produces a slowly progressive paralysis with spasticity without sensory symptoms; the spasticity is of the pure pyramidal type. This condition is considered by many neurologists to be nonexistent.

(2) *Hereditary spastic paraplegia* occurs in families; it appears in early youth as a slowly progressive paresis with spasticity. The chief lesion is sclerosis of the lateral cerebrospinal and posterior spinocerebellar tract, with slight sclerosis in the posterior column.

(3) *Syphilitic sclerosis of the cord.* In the case illustrated in Figures 170 and 206 the sclerosis is concentrated in the lateral cerebrospinal tracts, the pos-

terior spinocerebellar and rubrospinal tracts, with possible implication of the posterior spinothalamic tract and often with very little involvement of the posterior column. It gives symptoms of a bilateral upper motor neuron paralysis, with spasticity owing to the involvement of the neighboring rubrospinal tract; the vestibulospinal tract usually escapes. Sensory symptoms may be almost absent, and cerebellar symptoms are masked by the paralysis. The legs are usually first affected. The disease may last for several months only, ending in a myelitis or myelomalacia of weakened cord tissue; or it may run a course of extreme chronicity, slowly advancing through many years.

D. Simultaneous affection of upper and lower motor neurons.

Amytrophic lateral sclerosis (Figs. 186, 160 to 162). There is chronic degeneration of both lower and upper motor neurons affecting the lateral and ventral cerebrospinal tracts, and the anterior gray column cells of the cord. It occurs usually in adults. As a rule it begins in the cervical region of the cord and spreads upward to the motor nuclei of the medulla oblongata, causing one form of bulbar palsy. Where it appears in the cervical region, the arms may show typical lower motor neuron paralysis as the result of the loss of motor cells in the anterior gray columns, while the legs may be spastic owing to the involvement of the lateral cerebrospinal tracts in the cervical region.

E. Cord diseases affecting both motor and sensory tracts.

1. *Posterolateral or combined sclerosis* (Fig. 171) is associated with pernicious anæmia and with secondary anæmias. The sclerosis affects the posterior and lateral columns and is slowly progressive. Combined symptoms of tabes dorsalis and spastic paraplegia result; the sensory or paraplegic symptoms predominate according as the posterior or lateral columns are most affected. The severe pains and other sensory root symptoms of locomotor ataxia are absent, but there are sensory symptoms referable to the posterior columns and sometimes also to the posterior spinothalamic tract or to the posterolateral columns.

Similar scleroses are met with in cachetic conditions, pellagra, and ergot poisoning.

2. *Multiple cerebrospinal sclerosis* (Figs. 172 and 205). The disease is uncommon; it usually affects young adults and is probably due to infective multiple emboli. Multiple sclerotic lesions affect the white substance of the brain and cord; there are no secondary degenerations. The chief characteristics are paresis with spasticity, typical intention tremor (5 to 8 per sec.), slow monotonous scanning speech. Sensory symptoms are seldom marked.

F. Paralysis due to interruption of the sensory side of the reflex arc.

Experimentally in animals section of the posterior spinal nerve roots to a limb produces functional paralysis of the limb. The arm so affected is not used spontaneously, but may be used in coördination with the opposite arm. *In operative section* of the posterior nerve roots in man to relieve spasticity of the legs, no marked increase of paresis results if every third root be left.

In *tabes dorsalis* (Fig. 190) paresis is proportional to the affection of the

posterior nerve roots. Temporary paraplegic symptoms may follow tabetic crises.

Dejerine quotes cases (tubercle in pons involving medial lemniscus; subcortical abscess of parietal lobe) where profound loss of sensation on one side of the body caused loss of spontaneous use of the affected side. If the patients were asked to use the limbs, they could move them in an ataxic manner.

G. Reflex paralysis.

In painful joint affections the muscles waste, apparently through some reflex action in the cord segment involved. In long-standing cases there may be functional paralysis with contractures.

H. Paralysis without known lesions.

Familial periodic paralysis, of the flaccid type, occurs in youth after severe nervous strain. It disappears spontaneously, but tends to recur.

I. Hysterical paralysis.

May be hemiplegic, paraplegic, or monoplegic in type. Usually it is flaccid, rarely rigid; there may be contractures. The reflexes are seldom exaggerated; Babinski's sign is seldom present, the sphincters are seldom affected; there may be anomalous forms of anæsthesia and paræsthesia. It may last for a short time or for years; it usually yields to proper treatment for hysteria.

Lesions of Sensory Nerves or Tracts

Introduction

Affections of general sensation are either the only symptoms, or hold a prominent place in the symptom complex.

Anatomical and Physiological Data

Light touch. Before testing hairy parts must be shaved; the test is performed by a light pencil of cotton.

Lowest sensory neuron. The lowest sensory neurons form the cutaneous nerves in the posterior spinal nerve root and travel by two paths in the cord:

(*a*) They may go up the posterior column to the nucleus gracilis or cuneatus, to be relayed by a *second sensory neuron* through internal arcuate fibers to the opposite medial lemniscus which ends in the ventrolateral nucleus of the thalamus.

(*b*) After ascending 2 to 6 cm. in the posterior column, they may enter the posterior gray column of the cord and be relayed here by a *second senory neuron* which crosses in the gray commissure and joins the anterior spinothalamic tract to the ventrolateral nucleus of the thalamus.

From the ventrolateral nucleus of the thalamus the second sensory neuron is relayed by a *third sensory neuron* to the dorsolateral thalamic nucleus, where there is a redistribution of different forms of sensation. Thence impulses travel by a *fourth sensory neuron* to (*a*) and (*b*):

(*a*) To the essential thalamic organ (the medial thalamic nucleus) for representation in consciousness and for reactions of pleasure or discomfort.

(*b*) To the parietal cortex for tactile memories, comparisons and judgments, and to the anterior central and parietal cortex for voluntary motor response. Head and Holmes (*Lancet*, January, 1912) seem to regard the thalamocortical relay as arising in the essential thalamic organ.

Tactile discrimination. This is tested by a compass, of which the two blunt points are placed on the skin at the same time and the distance noted at which they are recognized as two. Also it may be tested by touching the skin with a blunt point such as a pencil point and asking the patient to indicate the point touched. The recognition of the point of contact is called topognosis (see Fig. 222).

First sensory neuron. Enters the cord with the cutaneous nerves, passes up the posterior column of the same side of the cord to the nucleus gracilis or nucleus cuneatus; it is relayed by the *second sensory neuron* by internal arcuate fibers to the formatio reticularis in the oblongata lateral to the medial lemniscus. The second sensory neuron joins the medial lemniscus near the thalamus and enters the ventrolateral nucleus of the thalamus where the stimulus is interpreted as touch without the power of judgment as to location or "twoness"; then it is relayed by a *third sensory neuron* to the dorsolateral thalamic nucleus and thence by a *fourth sensory neuron* to the parietal cortex for judgment of location and twoness, and judgments underlying the sense of form.

Pain, heat and cold.

1. *Lowest sensory neuron.* Pain from pin prick or from pinching the skin, *i.e.*, cutaneous pain, is carried by cutaneous nerves. Pain from deep pressure is carried by deep sensory nerves traveling with motor nerves. Heat and cold, or rather warm and cool between 22° and 42° C., or 72° and 104° F., are carried from specialized end organs, hot and cold spots, respectively, by cutaneous nerves. (Test by metallic tubes of warm and cool water.) More marked degrees of heat and cold above 50° C. and below 15° C., or 120° and 60° F., are carried by protopathic nerves in the skin.

2. *In the cord* all forms of heat and cold and all forms of pain are relayed a short distance above their entrance by second sensory neurons. The cells of these are in the posterior gray column, and the axons cross in the gray commissure and enter the posterior spinothalamic tract where, though close together, the tracts for each sense may be interrupted separately. They ascend in the oblongata and pons near the spinal tract of the trigeminal nerve, join the medial lemniscus in the mesencephalon, and so reach the ventrolateral nucleus of the thalamus.

3. From the ventrolateral nucleus of the thalamus they are relayed by the *third sensory neuron* to the dorsolateral thalamic nucleus and thence by a fourth *sensory neuron:*

(*a*) To the essential thalamic organ for pleasurable or painful reactions in consciousness.

(*b*) To the parietal cortex for memory records and comparative judg-

ments, and to the anterior central and parietal cortex for voluntary motor response.

[Note: Dejerine states that pressure pain is carried in the posterior columns homolaterally to the medulla oblongata.]

Conscious sense of position of limbs or fingers and of active or passive movements. To test the sense of active movements the patient is asked to touch his nose, ear, toe; to place fingers of both hands in accurate apposition; or to draw the heel along the opposite leg; all tests are performed with the eyes shut. For testing the sense of position the patient stands with heels together and eyes shut, or with eyes shut places his well hand on the affected limb or great toe after the physician has displaced it. For the sense of passive movement he is asked to indicate the angle through which a limb or digit has been displaced by the physician.

Lowest sensory neuron has sensory end organs in muscles, tendons and joints, travels deeply with motor nerves, ascends in the posterior column of the cord to the nucleus gracilis or nucleus cuneatus, and is relayed here by:

Second sensory neuron, which passes by internal arcuate fibers to opposite medial lemniscus by which these senses reach the ventrolateral nucleus of the thalamus. Thence they are relayed by a *third sensory neuron* to the dorsolateral thalamic nucleus and thence by a *fourth sensory neuron:*

(*a*) To the essential organ of the thalamus, where they reach consciousness and are associated with pleasurable or painful sensations.

(*b*) To the posterior central convolution for memories and judgments of position and movement, and probably thence to the superior and inferior parietal convolutions for tactile judgments of the form of objects. Also to the anterior central and posterior central gyri for voluntary motor response.

Sense of size and form in two or three dimensions is a sensory synthesis formed by combining judgments of the distance between points of contact on the skin and the sense of position of the limbs or fingers. The paths travel mainly in the posterior column, are relayed in the nucleus gracilis or cuneatus, cross and are again relayed in the ventrolateral nucleus of the thalamus, and are synthesized for judgments of size and form in the parietal cortex.

Pressure as tested by placing different weights on the supported limb is a form of deep sensation, travels up the cord by a bilateral path, *i.e.*, homolaterally by the posterior columns, and heterolaterally by the anterior spinothalamic tract after a relay in the posterior gray column.

This sense is relayed in the optic thalamus to the cerebral cortex for judgments of relative difference in pressure and is especially valuable in estimating lesions of the sensory cortex.

Sense of weight is tested by asking the patient, who must not be paralyzed, to estimate relative weights by raising them in each hand alternately. It reaches the thalamus through the posterior columns and medial lemniscus. It is especially valuable in testing cortical sensibility.

Sense of roughness is estimated by stroking the skin with a smooth instrument from which six blunt points may be protruded for measured distances (Graham Brown athesiometer). This test is especially useful in differentiating

thalamic and cortical lesions. A rough test may be made with sandpaper of different grades.

Sense of texture is tested by presenting the patient with different fabrics such as silk, cotton and velvet, to be identified by touch. The differentiation of these calls for sensory synthetic judgments and is especially valuable when used as a test for lesions of the sensory cortex.

Tuning-fork vibration is believed to be a peculiarly periosteal sense. It is elicited by placing the handle of a tuning fork (C fork with Gradenigo triangle) against a bony surface, and is a specially valuable means of testing conduction in the posterior columns in paralyzed patients. The path is by deeper peripheral sensory nerves through the homolateral posterior columns to the nucleus gracilis or cuneatus, thence across by the internal arcuate fibers to the opposite medial lemniscus and thus to the thalamus. It is little affected in cortical or thalamic lesions unless they are very gross.

Scraping the skin along the palms and soles, and tickling the palms and soles or hairy parts are valuable methods of eliciting the tendency to unpleasant oversusceptibility frequently associated with limited thalamic lesions. (For sensory tracts to the cerebellum, see later.)

Quality of sensory change produced by disease of sensory nerves and tracts. Irritation of sensory nerve endings or of ganglia on posterior nerve roots and certain thalamic lesions produce intense paroxysmal neuralgic pain. Certain cases of paroxysmal pain referred to the distribution of single nerves (for example, trifacial tic douloureux; sciatic neuralgia) have as yet been put on no definite pathological basis.

Irritation of posterior nerve root ganglia produces intense pain accompanied frequently by an herpetic eruption over the distribution of the nerve. The distribution of pain and rash is radicular in type.

Partial lesions of sensory conducting paths produce altered conduction which manifests itself as delay in time, heightening of the threshold so that a stronger stimulus is required to produce the characteristic reaction, and paræsthetic sensations of tingling, numbness, prickling, coldness, burning, or crawling accompanying ordinary unavoidable cutaneous stimuli. These sensations may be intensely disagreeable and are often interpreted as hyperæsthesia. Thus a pin prick may be delayed in conduction and need harder pressure to produce pain, yet be intensely disagreeable when it reaches consciousness, as though it exploded its way through an obstruction.

Total destruction of a conducting path is shown by complete loss of all forms of sensation normally passing along that route.

[Note: In describing the examination of a nervous subject the student should never use the terms "loss of epicritic sense, etc.," but should indicate definitely each variety of sense affected; thus, "loss or diminution of sense of pain by pin prick," loss of sense of warmth by test tube of slightly warm water," "cannot touch his nose with eyes closed," etc. Examination of a patient for sensory symptoms must be conducted in quiet surroundings; and should be divided into a series of short séances, continued at intervals, as such patients are easily fatigued and will then give unreliable answers.]

CLASSIFICATION

Lesions of the Sensory Neurons

A. Peripheral Nerve.

1. *Complete interruption* (Figs. 99 and 86).

(*a*) *Cutaneous nerve.* There is loss of sense of light touch, heat and cold between 72° and 104° F., tactile discrimination, and location of touch. Trophic changes may occur over the area of anatomical distribution of the nerve. (Exception: division of the radial nerve below its dorsal cutaneous branch in the arm is accompanied by only a very limited area of anæsthesia over the lateral side of the thumb; also the nerves of the trunk overlap each other to a considerable extent.) Stopford has lately shown that the cutaneous nerves to the fingers supply branches to the finger joints and that interruption of the digital cutaneous branches of the radial ulnar or median nerve is associated with more or less of sense of position in the affected finger.

(*b*) *Mixed nerve trunk.* The anæsthesia is the same as that just recorded for the cutaneous distribution of the nerve, with the addition of loss of pain and sense of pressure, loss of sense of heat above 120° F., and cold below 60° F., over a more limited area. There is also lower motor neuron paralysis and loss of sense of position and of movement of the muscles involved. There are trophic lesions of the skin. (See page 48, Fig. 86.)

Examples. Stab wound and gunshot wounds of nerves.

2. *Incomplete interruption* of a peripheral nerve causes diminished sensation and paræsthesia grading to anæsthesia of the skin and deeper tissues; there is also lower motor neuron paresis grading to paralysis of muscles involved according to the severity of the lesion. Hyperæsthesia and pain are variable (Fig. 210 and page 197).

B. Ganglia on Posterior Nerve Roots and Semilunar Ganglion on Trigeminal Nerve.

Herpes zoster, which is characterized by neuralgic pain along the distribution of the nerve root and therefore segmental in type, followed by a herpetic eruption, has been shown to be due to an inflammation of the ganglion on the posterior nerve root of the affected nerve or the ganglion on the fifth or seventh cranial nerve.

Similar pains and eruptions may follow pressure on the ganglion in tuberculosis or malignant tumors of the vertebræ.

C. Posterior Nerve Roots.

1. *Posterior radiculitis* (Dejerine, Figs. 208 and 191). This is usually of syphilitic origin; it may affect the posterior nerve roots only or the anterior and posterior nerve roots together. The lumbosacral and brachial regions are most often affected. The distribution of the pain, anæsthesia and lower motor

neuron paralysis is of radicular type and is confined to one side of the body. Reflexes are abolished for the muscles involved. Pain is increased by coughing and sneezing.

The anæsthesia may be total for all forms of sensation, or cutaneous sense may be lost and muscle sense retained. More rarely muscle sense is lost and cutaneous sense retained. Never are pain, heat or cold lost, and touch spared.

2. *Tabes dorsalis.* For the purpose of this study the underlying lesion in tabes dorsalis may be defined (Figs. 114 to 117, 190, 211 and 212) as a widespread chronic, slowly progressive syphilitic radiculitis, bilateral, affecting the posterior nerve roots, sometimes invading the root ganglia and causing secondary degeneration and sclerosis, best marked in the long neurons of the dorsal columns. All the symptoms of the disease are readily interpreted in terms of a slowly progressive interruption of the posterior nerve roots as is shown by a comparison with experimental section of these roots (page 144). The symptoms are as follows:

Lightning pains, and gastric, bladder, rectal and other crises due to irritation of the corresponding sensory nerve roots.

Herpes zoster occasionally, from invasion of the spinal ganglia.

Anæsthetic areas in skin, and general diminution of cutaneous sensibility.

Loss of visceral sensory impulses with consequent functional disturbances of the bowel bladder or genital sense.

Loss of muscle sense with consequent ataxia, atonia, and later paresis.

Loss of reflexes.

Trophic lesions, such as perforating ulcer of foot and Charcot's joint (Figs. 19 and 120).

All these are the symptoms to be expected from the nature of the lesion. The anæsthesia is radicular in type (Fig. 118). In early tabes the commonest sites of cutaneous anæsthesia are the medial side of the arms, or a girdle round the upper thorax, or, more rarely, the lateral side and back of the thigh or the perineum.

Later, loss of muscle sense, the sense of position, and the sense of motion are most marked, giving rise to ataxia.

Also there is loss of tuning-fork and compass sense (Figs. 117, 118, 211 and 212). In fact, the long posterior neurons of the first order suffer most. (See introduction to sensory classification.) The secondary sensory neurons are not involved. (See general discussion of table dorsalis on page 199.)

D. Sensory Tracts in Spinal Cord and Brain Stem.

1. *Primary posterior sclerosis* such as Thompson's case of partial posterior sclerosis (*Brain*, Vol. 34, p. 510). (See Fig. 103 and page 203.) Such sclerosis is usually associated with anæmia; it is limited to parts of the posterior white columns.

The leading symptoms in Thompson's case were a sense of deadness, fullness, and tingling; *no pain.* There was loss of sense of passive position and consequent ataxia, the sense of weight was impaired. Tactile discrimination was lost and

localization poor. Tuning-fork sense was not tested but was probably lost. There was no loss of pain, heat, or cold, or of tactile sense.

The essential difference between this and tabes dorsalis is that tabes dorsalis is primarily a posterior nerve root lesion; while this is a tract lesion from the beginning. The leading symptomatic differences are a more acute course and an absence of pain in primary posterior sclerosis.

2. *Posterior spinothalamic tracts. Operative section* of the posterior spinothalamic tract has been successfully done for the relief of pain in pelvic cancer. One case showed loss of the sense of pain; heat was felt as cold; there was no other sensory disturbance and no motor loss. (See page 151, Martin and Spiller's case, Jour. Am. Med. Assn., May 18, 1912; Fig. 85.)

A *tuberculous focus* interrupting the posterior spinothalamic tracts was described by Spiller in Penn Med. Bulletin, July and August, 1905. (See page 151; Fig. 104.) There was loss of pain and of temperature sense and no other sensory loss.

3. *The anterior spinothalamic tract.* As simple touch and pressure are conducted both by this tract after crossing and by the dorsal columns homolaterally, sclerosis or other interruption of this tract alone would cause no symptoms.

4. *Lesions of the second sensory neuron for pain, heat, and cold in the spinal gray matter or anterior commissure. Syringomyelia* (spinal gliosis, Figs. 187, 188, 163, 164, 215 and 216). This is a slowly progressive overgrowth of central neuroglia accompanied by cavity formation; it is most common in the lower cervical segments or medulla oblongata, but is possible anywhere in the cord.

In its early stages it invades the parts about the central canal and spreads into one or both posterior gray columns. It may thus destroy the nerve cells or tract of the second sensory neuron for pain, heat and cold in the posterior gray column or commissure; other parts of the cord may at first escape (see Figs. 224, b, 9 and 9′). Therefore, the earliest symptom is loss of pain, heat and cold of segmental distribution, over one side if the posterior gray column be the chief site of the lesion, bilaterally if the commissure suffer (Fig. 215). As tactile sense travels by the homolateral posterior column as well as by a crossed path, tactile sense escapes. The typical early sign of syringomyelia is loss of pain, heat and cold with retention of sense of touch over a segmental sensory area. Later the anterior gray column may be invaded, causing a segmental lower motor neuron paralysis with wasting, R. D. (Fig. 216), and fibrillary twitchings. If the posterolateral column be destroyed there will still be a lower sensory loss for pain, heat and cold, and trophic symptoms of segmental distribution; and as the disease advances the symptoms may assume the Brown-Sequard type (Fig. 214) or those of a complete transverse lesion (Fig. 217).

Hematomyelia. If hemorrhage into the spinal cord, traumatic or idiopathic (Fig. 213), be into the central canal and gray matter, it may simulate syringomyelia, but the onset is acute.

[Note: *Syringobulbia* or *gliosis* of the floor of the fourth ventricle manifests itself usually as an invasion of the twelfth or of the sixth nucleus with a lower motor neuron paralysis of the twelfth or sixth cranial nerve. It may involve

the fasciculus solitarius with homolateral loss of taste, or the roots of the seventh, vagus, or accessory nerves. It thus constitutes one form of bulbar paralysis, the usual causes of which are polioencephalitis, bulbar amyotrophic lateral sclerosis, and bulbar gliosis.]

Upper cervical cord. A gliosis affecting the posterior gray column of the cord and the spinal tract of the trigeminal nerve in the second and third cervical segments may cause a second sensory neuron loss of pain, heat and cold for the distribution of the second and third cervical nerves (segments) on the side of the lesion owing to destruction of the posterior horn cells which relay the second and third nerves to the opposite spinothalamic tracts; touch escapes. The face may suffer a first or second sensory neuron loss of touch, pain, heat and cold for part of the face (segmental facial distribution) owing to destruction of the substantia gelatinosa relaying the spinal tract of the fifth nerve to the opposite trigeminothalamic tract.

5. *Oblongata. Lesion of the reticularis grisea in the medulla oblongata* (Figs. 176 and 177).

In the oblongata occlusion of the posterior inferior cerebellar artery (Fig. 176) may cause softening of the spinal tract of the fifth nerve with the neighboring spinothalamic tract. The result is loss of pain, heat and cold for the opposite side of the body and neck; touch and muscle sense and the sense of position, etc., which travel by the medial lemniscus, escape.

There will be general sensory loss for the distribution of the fifth nerve (face, conjunctiva, tongue) on the same side as the lesion. There may be no other symptoms, or the glosso-pharyngeal, or vagus roots or nuclei, or the restiform body may be involved with homolateral loss of taste, pharyngeal and laryngeal paresis and cerebellar symptoms.

6. *Medial lemniscus.* Thrombosis of one anterior spinal artery near its origin may cause softening of the medial lemniscus and part of the pyramid in the medulla oblongata; the hypoglossal nerve is usually also involved (Fig. 174). A lemniscus lesion here causes loss of sense of position, and of passive and active motion, and consequent ataxia, and loss of tuning-fork sense in the opposite side of the body (not the face).

Pain, heat and cold, and touch travel upward near the spinal tract of the fifth nerve; they escape. Tactile discrimination does not run in the medial lemniscus here and also escapes.

There will be a lower motor neuron paralysis of the tongue on the same side, and a spastic paralysis of the opposite side of the body from involvement of the pyramidal tract above the decussation.

Many other bulbar combinations are possible. (See Figs. 175, 176 and 177.)

7. *Tegmental lesion in lower pons,* as by hemorrhage into the pons or tubercle in it (see Fig. 179).

Such a lesion may destroy Deiters' nucleus, the seventh nucleus and nerve, the sixth nerve, the spinal tract of the fifth nerve, the spinothalamic and trigeminothalamic tracts, the rubrospinal and vestibulospinal tracts. In the case recorded the sixth nucleus, the medial lemniscus, the corpus restiforme and basillary portion of the pons escape.

Sensory Symptoms

(Fig. 179)

Structure destroyed	*Symptoms*
Spinal tract of fifth nerve	Homolateral anæsthesia of face, all forms.
Trigeminothalamic tract	Loss of pain, heat, and cold for face on side opposite to lesion.
Posterior spinothalamic tract	Loss of pain, heat, and cold for opposite side of body. Touch, muscle sense, tuning-fork sense, which travel by medial lemniscus, escape.

Motor Symptoms

Structure destroyed	*Symptoms*
Sixth nerve	L. M. N. paralysis of the homolateral lateral rectus; eye is turned inward by unopposed action of medial rectus.
Seventh nerve	L. M. N. paralysis of same side of whole face. Eyelid cannot be closed, angle of mouth droops, mouth drawn to opposite side. Furrows of brow and face are lost.
Destruction of Deiters' nucleus with loss of vestibular fibers to homolateral sixth nucleus and heterolateral medial longitudinal bundle.	Destruction of vestibular mechanism for turning both eyes to the side of the lesion; therefore the opposite eye is turned outward owing to enfeebled action of medial rectus. If the sixth nerve had escaped it would have been found to act feebly for the same reason.
Destruction of vestibulospinal tract	Should cause loss of muscle tone on the same side of the body.
Destruction of rubrospinal tract	Should cause homolateral tremors and cerebellar ataxia.

As the medial lemniscus escapes, there is no loss of tactile, tuning-fork, or muscle sense, and no ataxia. As the pyramidal tract escapes, there is no hemiplegia. Other pontine combinations with sensory loss are illustrated in Figure 178, a and b.

8. *Mesencephalic tegmental lesions* (Fig. 181). The nearer one approaches the thalamus, the more closely are all forms of sensation for the whole of the opposite side of the body, face and oral and nasal mucous membranes grouped together in the medial lemniscus. Therefore, a tegmental lesion in the mesencephalon or a lesion in the subthalamic region involving the medial lemniscus will cause anæsthesia for all forms of sensation of the whole of the opposite side of the body and face, and the nasal, oral and ocular mucosæ. If the anæsthesia be profound, there will be functional paralysis of the anæsthetic side of the body,

though if the basilar portion including the pyramidal tract escapes, the parts may be used when the patient is told to use them.

If the red nucleus be involved there will be heterolateral tremors, though the sensory loss and sensory ataxia may prevent the appearance of these and other cerebellar symptoms.

As each ear is represented bilaterally in the higher centers, no one-sided lesion of the second auditory neuron (lateral lemniscus) causes deafness of either ear.

Destruction of the third nucleus or fila causes homolateral paralysis of all the eye muscles supplied by the third nerve, the pupillary and ciliary fibers sometimes escaping.

9. *Thalamic lesions.* If one includes with the thalamus the medial and lateral geniculate bodies, then all forms of sensation are represented in the thalamus with the possible exception of the vestibular equilibratory sense.

For the present purpose it is preferable to exempt the lateral geniculate body and pulvinar, whose function is visual, the medial geniculate body whose function is auditory, and the anterior nucleus of the thalamus whose function is olfactory.

The medial nucleus of the thalamus is of unknown function unless it be the essential thalamic organ; if so, it is the organ of elementary sensations with an affective reaction of pleasure and pain.

There remains the *lateral nucleus of the thalamus* (Fig. 87). This is divisible into a ventral and a dorsal portion and these again into several nuclei. In the ventrolateral nucleus all forms of bodily sense, including visceral sense, are relayed, and all are crossed with reference to their point of peripheral origin. All thalamic symptoms are referred to the side of the body opposite the lesion.

From the ventrolateral nucleus all forms of sensation, with the possible exception of the sense of position, are relayed to the dorsolateral nucleus and thence to the essential organ of the thalamus (Head and Holmes, *Lancet*, January, 1912) which may be in the medial nucleus, where they reach consciousness and are associated with pleasurable or unpleasant "feeling tone." This essential thalamic organ is under cortical control by virtue of thalamocortical and cortico-thalamic fibers radiating through the internal capsule between the dorsolateral thalamic nucleus and the cerebral cortex; the thalamocortical fibers are mainly distributed to the anterior central and parietal convolutions, the cortico-thalamic come from perhaps all parts of the cortex. This cortical control inhibits thalamic reactions to pleasure and pain. These thalamocortical relays also underlie sensory memories, comparisons, judgments, and voluntary motor responses.

There are also thalamocaudate, pallidothalamic, and cerebellothalamic connections, the first two homolateral and the last crossed, and each of these may have motor reactions through the cerebral cortex.

Thalamic lesions. Most thalamic lesions are vascular, either hemorrhagic or ischæmic; the latter are due to thrombosis or embolism of the basilar arteries and cause softening.

Gross lesions if not fatal cause profound sensory loss for all forms of sensation and may be indistinguishable from similar lesions in the subthalamic region. If the internal capsule be involved there will be concurrent hemiplegia, and there will be hemianopsia if the optic radiation or pulvinar(?) be included.

Limited thalamic lesions may involve almost any form of sensory loss to the exclusion of others.

Summary of Sensory Loss in Limited Thalamic Lesions
(Head and Holmes, *Lancet*, 1912)

Sense of posture and of recognition of passive movements is most frequently affected. The degree varies greatly, from slight diminution to complete loss.

Pain sense. While the threshold may be raised, painful sensations when felt are intensely disagreeable and the reactions are uncontrollable.

Tactile sense is frequently diminished, seldom abolished; it is frequently confused by a sensation of tingling.

Localization depends on tactile sense; it is defective in half the recorded cases where tactile sense is preserved.

Compass sense, where it is possible to elicit it, shows widening between the points of contact which are recognized as two.

Thermic sense. In twenty-two out of twenty-four cases there was excessive response to the unpleasant character of heat and cold. The threshold may be raised or normal; or there may be complete insensibility of both heat and cold; or discrimination between heat and cold for temperatures above 40° C. or below 26° C. may be lost, both being called either hot or cold.

Sense of weight depends on the sense of posture and movement, and varies with those senses.

The *appreciation of size, shape and form* vary with tactile and postural sense.

Vibration sense is lost, or at least duration of the sensation is usually shortened.

The *sense of roughness* is not much affected.

The Thalamic Syndrome of Dejerine (Fig. 220) is found in limited lesions of the dorsolateral thalamic nuclei according to Head and Holmes; or of the posterior part of the ventrolateral thalamic nucleus according to Dejerine. It consists in the following characteristic grouping of symptoms, all referred to the opposite side of the body:

(*a*) A persistent loss of superficial sensation—touch, pain, heat and cold for the opposite side of the body, varying in intensity.

(*b*) A greater loss of deep sensibility (pressure, muscle sense).

(*c*) Slight hemiataxia.

(*d*) More or less loss of sense of size, shape and form in three dimensions.

(*e*) Acute pains, persistent, paroxysmal, often intolerable, yielding to no analgesic.

(*f*) Slight hemiplegia without contractures, and passing off rapidly.

(*g*) Choreic and athetoid movements. To these add the Head and Holmes symptom:

(*h*) A tendency to react excessively to unpleasant stimuli.

10. *Sensory loss in lesions of the internal capsule.* The usual lesions are vascular, causing softening of or hemorrhage into the capsule. The thalamus may or may not escape.

Anatomical considerations (Fig. 87, e). By the time the internal capsule is

fairly differentiated into an anterior limb, knee and posterior limb, the only purely sensory part of the capsule is the retrolenticular portion. This is chiefly occupied by the optic radiation (geniculo-calcarine and thalamo-calcarine fibers). Lesions of this tract cause homonymous hemianopsia, that is, blindness of the homolateral half of each retina and of the opposite half of each visual field.

In the knee and posterior limb between the thalamus and lentiform nucleus the cortical motor fibers are mixed with thalamocortical fibers which relay the sensory centers in the thalamus to the sensory portions of the cerebral cortex. Therefore, lesions here, in addition to the resulting hemiplegia, cause sensory loss of cortical type and capsular distribution, that is, affecting the opposite half of the face and trunk and opposite limbs (see capsular motor lesions, page 237, and lesions of the sensory cortex, page 214). The sensory relays for smell and taste are too remote to be affected. The auditory radiation is usually too near the base to be affected; also each ear is bilaterally represented in the cortex (Figs. 218, 219 and 222).

Sensory judgments of tactile localization and discrimination, judgments of position, weight, form and texture, suffer most. Primary senses of pain, temperature and touch usually escape. Hands, feet and tongue, where touch is most highly educated, are most affected.

11. *Lesions of the sensory cortex* (Figs. 142 and 143). *Location.* The following statement is probably correct (Figs. 142 and 143): Touch and sense of movement and posture are primarily represented in the posterior central gyrus; the sensory areas lie more or less closely behind the motor areas in the anterior central gyrus, though the hand and foot, especially the former, have probably the widest representation owing to their greater education.

Sensory memories underlying judgments of size and form are in the immediate neighborhood of the anterior end of the intraparietal sulcus (Fig. 142). Here lies the center for stereognostic sense (tactile gnosia).

Common lesions of the sensory cortex are traumatism, arteriosclerosis with thrombosis, or embolism, and neoplasms.

No stationary cortical lesion causes complete loss of the primary forms of sensation, and when the lesion is progressive the completeness of the loss is due to diaschisis (shock or exhaustion, producing temporary separation of synapses).

The sensory loss due to cortical lesions is always of the nature of defective powers of comparison with previous memories, and the defective formation of sensory judgments (Fig. 221). As a rule all forms of sensory loss are most marked at the extremities of the affected limbs, and decrease from below upward. The distribution of cortical anæsthesia, like cortical paralysis, is of the monoplegic type. Ataxia of the affected limb is proportional to the loss of the sense of position and motion.

In cortical sensory lesions *responses to tactile stimuli* are characterized chiefly by inaccuracy, uncertainty and variability of replies on examination, accompanied by sensory hallucinations.

Simple touch on hair-covered parts is never lost.

Pain sense is unaltered.

Temperature sense is unaltered except for diminished powers of comparison.

Estimation of posture of the limbs or digits and passive movements are most affected.

Localizing power of spots touched is not commonly lost but becomes uncertain.

Compass test does not always show loss of sense of twoness, but the distance is often increased. The sense of twoness may be lost.

Estimation of different weights of objects of the same size and shape is frequently disturbed.

Appreciation of size, shape and form is usually markedly affected.

Sense of roughness is not lost but comparisons leading to the recognition of material (silk, cotton, wool, velvet) by touch is lost.

Tuning-fork vibration is not lost, but may be less plain.

Patients suffering lesions of the sensory cortex are easily exhausted during examination on the affected side, and replies readily become uncertain and accompanied by sensory hallucinations while remaining accurate for the unaffected side.

A lesion of the sensory cortex may cause Jacksonian epilepsy preceded by a sensory aura.

CEREBELLUM

Anatomical Considerations

(Figs. 121 and 128)

The *flocculus* is rudimentary in man.

The *vermis* and *hemispheres* are each independent and there are no commissural or association fibers between them. In the vermis the nuclei fastigii and globosi are older phylogenetically than the cortex, while the vermis is older than the hemispheres.

Afferents to vermis. From all the muscles of the body, especially of the same side, afferent fibers to the vermis pass by the posterior spinocerebellar tract and restiform body and by the anterior spinocerebellar tract through the superior medullary velum; collaterals pass to the nucleus dentatus from the restiform body. Afferents to the vermis also come from the optic and auditory nerves.

Efferents. From the cortex of the vermis efferents pass to the nuclei fastigii and globosi, mainly homolaterally; perhaps also to the dentate nucleus. From the nuclei fastigii and globosi efferents go to Deiters' nucleus and to the nuclei of the spinal tracts of the vestibular nerves and almost certainly directly to the spinal cord joining the vestibulospinal tract; these are mainly homolateral.

Action. The vermis acts on all lower motor neurons, but especially on those required for simultaneous bilateral action (eyes, head, neck, trunk, standing, walking, phonation, speech).

Hemisphere. The cerebellar hemisphere appears first in birds and develops with the cerebral cortex; each is independent of the other and of the vermis. Each acts on all muscles, mainly homolaterally; probably in man each acts mainly on the arms, and on the legs for more specialized movements.

Afferents. From the cerebral cortex, mainly the anterior and posterior central

convolutions and middle temporal gyrus (vestibular cortical center) of the opposite side fibers pass by the basis pedunculi to the pons; they are interrupted in the nuclei pontis and pass thence to the opposite cerebellar cortex by the brachium pontis.

There is a probable afferent tract for the eyes from the occipital cortex by the pons or from the superior colliculi through the brachium conjunctivum to the nucleus dentatus and the cortex.

Afferents go from body muscles by spinal endogenous neurons; and from subcortical ganglia, by the central tegmental tract; both go to the inferior olive and thus through olivocerebellar fibers to the opposite cerebellar cortex.

Efferents. Efferent fibers go from the cortex to the nucleus dentatus, thence by the brachium conjunctivum to the opposite red nucleus and optic thalamus; the optic thalamus is connected directly and by a rubrothalamic connection. The influence of the efferents may end in the optic thalamus, modifying thalamic impulses and thus indirectly acting on the motor mechanism; or it may reach the motor cortex by a special thalamocortical relay; or it may reach lower motor neurons through the rubrospinal tract. All efferent connections are overwhelmingly homolateral.

Functions. The cerebellum is the head ganglion of the proprioceptive system (Sherrington).

Cerebellar lesions produce no defect in conscious sensation. (Horsley, Thomas, Holmes).

The cerebellum is not especially an organ of equilibration (Holmes).

The cerebellum receives and interprets proprioceptive impulses from all parts of the body, and by virtue of these keeps the motor mechanisms in such a state of tone that they can respond promptly and efficiently to voluntary impulses; it thus assures the correct coöperation of the separate motor centers that are concerned in individual acts (Holmes).

Add to this that in the absence of the cerebral cortex in experimental dogs the cerebellum has a similar regulating influence on lower cerebral motor centers (corpora striata or red nucleus through the optic thalamus) and on the spinal motor mechanism, so that dogs without the cerebral cortex can still perform all automatic movements apparently normally, and only (1) memories, (2) judgment, and (3) volition are wanting.

Arguing from this, it seems a legitimate working hypothesis that in man the cord plus the optic thalamus plus the corpora striata, regulated by the cerebellum may take care of acquired automatisms (walking, balancing, typing, piano-playing), thus relieving the cerebral cortex of all except initiatory and inhibitory influences for acquired automatisms. In this way the cerebral cortex has more energy available for higher purposes.

It is probable that the cerebellar nuclei are capable of considerable action independently of the cerebellar cortex.

In the absence of one cerebellar hemisphere almost complete compensation may in time be effected through the opposite cerebral hemisphere (and remaining cerebellum(?)). Considerable cerebral compensation is possible in dogs with complete absence of the cerebellum (Figs. 122 to 127).

Symptoms (Figs. 128A to 132). Congenital absence of the cerebellum is attended by symptoms which are proportionately very small when compared with the importance of the cerebellum in adults. Slowly forming cerebellar abscess in one hemisphere or slowly developing cerebellar cirrhosis may reach a very advanced stage without symptoms, presumably owing to progressive compensation.

The symptoms of cerebellar lesions are bilateral if the vermis is affected; homolateral in proportion as the lesion is one-sided.

Vertigo appears early in acute lesions; it is soon lost; it may be due to irritation of the vestibular apparatus(?).

Diaschisis is rarely obvious.

Forced rotary movements rarely occur in man, and then only immediately after operation or severe injury; they have been seen in laceration of the brachium pontis (Starr). Patients suffering from acute lesions tend at first to lie on the affected side (on healthy side, Stewart and Holmes).

Atonia appears early and lasts long. The muscles feel soft; all the limbs feel "floppier" to the patient than normal limbs.

Voluntary movements show:

1. Asthænia; lack of normal power in movements that require exertion. Muscles tire easily.
2. Slowness in initiation and stoppage of each movement.
3. Discontinuity and irregularity in maintenance of muscular contractions.
4. Ataxia of cerebellar type. Cerebellar ataxia can be analyzed into the atonia, asthænia, slowness, and discontinuity of movement with the addition of:

   (*a*) Asynergia—lack of coördinated action between agonistic antagonistic and synergic muscles.
   (*b*) Dysmetria—lack of perfect adjustment of each individual movement to the required purpose.
   (*c*) Decomposition of movement (Holmes)—resolution of a compound movement into its elements.
   (*d*) Deviation from the line of movement, usually toward the side of the lesion, early compensated for with a tendency to overcorrection.
   (*e*) Adiadokokinesis—difficulty in producing reverse movements rapidly in succession (pronation and supination; making and breaking the fist; scissors movement; erasing with rubber).
   (*f*) Static intention tremor—slow, irregular, especially noticeable when the muscles are tired; slow failure; jerky return.
   (*g*) Spontaneous deviation in Bárány's pointing test—deviation of affected hand toward the side of the lesion.

Abnormal attitudes. The occiput is tilted toward the homolateral shoulder; this is an early symptom, only seen in acute cases. The trunk is bent with the

concavity to the side of the lesion. Where the lesion is in the middle of the vermis, the head is bent backward.

In vermis cases the face is stolid and expressionless.

The standing attitude is stiff and rigid, body concave to the homolateral side, foot abducted and rotated outward; most of the weight is placed on the opposite leg. The patient can balance on the opposite leg, but not on the homolateral one. The homolateral leg is apt to give way, but the patient can stand securely with eyes closed.

Gait is like that of a drunken man, with a tendency to fall to the affected side. There is a wide base of support; the homolateral leg is abducted, and moves disjointedly; the line of progression is deviated toward the affected side at first; later the patient may overcorrect this tendency. The affected arm does not swing naturally in walking. The patient may tend to fall backward from failure to bring the trunk forward with the advancing legs.

Eye symptoms:

1. Skew deviation (Fig. 125) occurs only in early stages of acute cases. It is an asthenic deviation of the eyes; the homolateral eye turns downward and inward, the opposite eye upward and outward.
2. Nystagmus occurs on attempts at fixation, especially on extreme fixation toward the side of the lesion. There is slow failure and quick, jerky return. This is a very constant symptom in acute cases and persists for a long time, especially in attempts at extreme deviation toward the side of the lesion.

Speech. After recent and severe injuries speech is slow, drawling, monotonous; it tends to be staccato, scanning, singsong, difficult to understand, often explosive, and is accompanied by evident effort and by facial contortions. It improves rapidly after gunshot injuries, but is more or less affected for months.

Knee jerk. On the homolateral side the knee jerk is feebler, less brisk, and less easily elicited than on the other side; it tends to the pendulum quality.

Cerebellar convulsions are a rare accompaniment of cerebellar tumors, especially tumors of the vermis or of the cerebello-pontine angle. The exact cause is not understood. They are tonic in character, thus contrasting with cerebral convulsions which are clonic. In vermis tumors the head is arched backward, the back is arched, the hands are supinated, the elbows flexed, the arms adducted, and the legs and ankles are in full extension.

In hemisphere and cerebello-pontine angle tumors the tonic contractions are more marked on the affected side. The eyes are spasmodically deviated from the side of the lesion. (Dana, N. Y. Med. Jour., 1905; Stewart and Holmes, *Brain,* 1904; Cushing, *Tumors of the Nervus Acusticus,* 1916.)

To the foregoing symptoms, due to the injury to the cerebellum itself, must be added:

1. General symptoms in cases of cerebellar tumor. These are choked disk, headache, vomiting, convulsions if the aqueductus cerebri be obstructed; attacks of unconsciousness occurring for the same reason.

2. Neighborhood pressure symptoms. There may be paresis or paralysis of the muscles supplied by fifth nerve, seventh nerve, glossopharyngeal-vago-accessory group; affections of hearing (cochlear nerve); vestibular symptoms (vestibular nerve, Deiters' nucleus); more rarely third, fourth, sixth, or twelfth nerve symptoms.

Compression of pyramidal tract may be present, causing spastic hemiplegia; or there may be compression of the nucleus gracilis or nucleus cuneatus with posterior column symptoms.

Irritation of the floor of the medulla oblongata may cause polyuria, glycosuria, and polydypsia.

Diseases of the Cerebellum

A. Of the cerebellum itself.

1. *Agenesis and hypoplasia.* Total or partial absence of the cerebellum may occur, or the cerebellum may be congenitally small though apparently normal in other respects. Some cases have shown surprisingly few symptoms, and the condition has not been suspected during life. Other patients learn to walk very late and with much difficulty, are unsteady in their movements, are easily fatigued, and may show poor mental development.

2. *Hereditary cerebellar ataxia* (Marie, Sanger-Brown). This appears after the twentieth year. There is degeneration mainly in the cerebellum, less marked in the cerebellar tracts of the spinal cord.

3. *Familial spinocerebellar ataxia* (Friedreich's, Fig. 189). This disease appears late in childhood in several members of the same family. It involves the spinal cord more than the cerebellum; the spinocerebellar tracts are most affected, next in order the posterior columns and the cerebrospinal tracts.

Two and three are so closely related that they are classed here together. They appear to be due to hereditary weakness of the cerebellar system. The symptoms are mainly cerebellar with the addition of posterior column symptoms ataxia of the tabetic type), and upper motor neuron symptoms (spastic paraparesis), varying with the distribution of the lesion.

4. *Slowly progressive cirrhosis or atrophy* may occur (Figs. 130 and 131), including cirrhosis of the inferior olives, the gray matter of the pons, and the middle peduncle. The corpora dentata and other cerebellar nuclei with the brachia conjunctiva may escape. Some cases show few symptoms; while others show fairly typical cerebellar atonia, asthenia, ataxia, adiadokokineses (gait, standing posture, and voluntary movements all affected); eye symptoms are seldom present.

5. *Abscess in one hemisphere if slowly progressive* and especially if it does not invade the corpus dentatum may be practically without focal symptoms.

6. *Intracerebellar tumor* if it is situated in the *vermis* shows rapidly progressive symptoms, both local symptoms of a focal character and remote symptoms due to pressure.

If the tumor be in a hemisphere and growing slowly, it may become large without giving rise to symptoms. When these appear, they are mainly homo-

lateral. In more rapidly growing tumors the general symptoms appear early (headache, vomiting, choked dick, blue blindness); and the focal symptoms are well marked.

7. *Cerebellar hemorrhages and softenings* are so frequently complicated by similar lesions in other parts of the brain that a distinctive symptomatology has not been established.

8. *Traumatism.* Gunshot wounds when not immediately fatal show a more or less typical cerebellar syndrome as enumerated in the general symptomatology (Gordon Holmes, *Brain,* 1917).

B. Affections of the afferent cerebellar paths.

1. *Friedreich's spinocerebellar ataxia* (Fig. 189), already mentioned, primarily and chiefly affects the posterior spinocerebellar tracts in late childhood. The chief cerebellar symptoms are cerebellar ataxia, asthenia, and atonia. These are mixed with posterior column and upper motor neuron symptoms. The disease is bilateral.

2. *Softening or tubercle or other localized lesions in the restiform body* (Fig. 129). These may give typical homolateral cerebellar atonia, asthenia, ataxia, adiadokokinesis. The trunk and legs (standing, walking) suffer most. The chief neighborhood symptoms would probably be due to lesions of the spinal tract of the trigeminal, and of Deiters' nucleus.

3. *Brachium pontis.* Starr records a case of laceration of the brachium pontis in fracture of the base; the patient had forced rotatory movements toward the side of the lesion till he died three days later. (Was the vestibule or vestibular nerve or nucleus also injured?)

Softening, tubercle, gumma, etc., of the brachium pontis give typical lateral hemisphere symptoms.

[Note: Any extensive lesion of the basilar part of the pons should theoretically produce bilateral cerebellar symptoms because of interruption of the cerebro-ponto-cerebellar afferent pathways of both sides. I have found no record of such symptoms, partly perhaps because the hemiplegia overshadows other defects. In these cases evidences of cerebellar lesion should be sought.]

C. Lesions of the chief cerebellar efferent pathway.

1. A lesion of the *corpus dentatum* causes homolateral symptoms of marked severity, as all cortical efferent fibers from the corresponding hemisphere pass through it.

2. A lesion of the *brachium conjunctivum* in the mesencephalon will almost certainly involve the lateral and medial lemniscus, the rubrospinal tract and perhaps the basis pedunculi, as well as the nuclei or roots of the homolateral third and fourth nerves. The cerebellar and lower motor neuron symptoms are homolateral; the lemniscus and basilar symptoms are crossed.

The chief cerebellar symptoms which are distinguishable from those due to the neighboring tract and nerve lesions and are not too much masked by them,

are nystagmus and homolateral intention tremor. For the symptom complex, see mesencephalic lesions.

The lesions of *disseminated sclerosis* may invade the corpus restiforme, brachium pontis, cerebellar nuclei, or brachium conjunctivum, thus adding cerebellar symptoms to the other symptoms of multiple sclerosis. The typical intention tremors of disseminated sclerosis are probably cerebellar in origin.

D. The inferior cerebello-pontine angle.

Juxtacerebellar tumor, usually neuroma of the acoustic nerve, is frequent in the inferior cerebello-pontine angle, comprising 6% of all brain tumors (Cushing, *Tumors of the Nervus Acusticus,* 1917). The first symptoms are usually due to irritation or paralysis of the cochlear and vestibular nerves—tinnitus and deafness, both homolateral; nystagmus and vertigo. There is paroxysmal pain occipital or frontal; with these there are fluctuating symptoms, referable to neighboring cranial nerves (fifth, sixth, seventh, less often the glossopharyngeal, vagus, and accessory). Later unsteadiness of gait and other homolateral cerebellar symptoms are added owing to pressure on the corpus restiforme, brachium pontis, or cerebellar hemisphere.

Such tumors may be successfully removed and hence early diagnosis is important. They are usually slow growing, are rare before thirty years of age, and seldom come to operation in less than four years from the first appearance of symptoms.

Affections of the Corpora Striata and Red Nuclei

General symptoms. The most prominent symptom of disease of the corpora striata and red nuclei is the occurrence of *constant slow tremors,* two to four per second, which stop during sleep and are usually lessened during voluntary movement.

Rigidity, usually general, affects the flexors and extensors alike. Voluntary movement is not abolished, but is often made difficult by this rigidity of the muscles. There is no Babinski and no exaggeration of tendon reflexes, but they may be difficult to elicit owing to the equal rigidity of the opposed muscles; the abdominal reflexes are present.

1. *Lenticular degeneration.*

   (*a*) *Subacute familial type* (Figs. 133 to 136) associated with cirrhosis of the liver, occurs in young adults and in two or three members of the same family, but is not hereditary; patients die in two to seven years.

   (*b*) *Chronic type,* without cirrhosis of liver, commences in early life and is slowly progressive, but may last for many years.

2. *Paralysis agitans or Parkinson's disease* is rare before the age of forty years (Fig. 137). There is chronic slowly progressive tremor and rigidity, with a characteristic running gait in a stooped position. Evidence is accumulating to show that this is due to degeneration in the globus pallidus or in the substantia nigra.

3. *Huntingdon's Chorea.* This is of a hereditary character; it appears in adult life and is accompanied by a tendency to mental deterioration and dementia. It is slowly progressive; there is some evidence that it is due to degeneration of the caudate nucleus and putamen. Possibly athetosis may have a similar cause. (See Ramsay Hunt, "Progressive Atrophy of the Globus Pallidus," *Brain,* 1917.)

Chorea major, hysteria major, and torsion spasm may be due to simultaneous disease of the caudate nuclei and the cerebral cortex.

Many cases of lethargic encephalitis are followed by the Parkinsonian syndrome, chorea, choreoathetosis or torsion spasm according to whether the neostriatum or palæostriatum is more affected.

Mixed Lesions of the Motor and Sensory Tracts

1. *Posterolateral or combined sclerosis* (Fig. 171). Degeneration of the posterior and lateral columns in various combinations may be associated with severe anæmias and cachexias; the disease lasts from one to five years.

Symptoms. Spastic paresis due to the involvement of the lateral cerebrospinal tracts combined with atonia, ataxia, and loss of knee jerks due to the posterior column lesion. The succession of symptoms varies with the order of involvement of the posterior or lateral column.

The only sensory symptoms are paræsthesia and loss of muscle, compass, and bone sense.

The severe pains of locomotor ataxia are absent, as the nerve roots are not affected.

2. *Syphilitic combined sclerosis.* Some cases of syphilis of the spinal cord appear to follow a combined type.

3. *Friedreich's ataxia or familial spinocerebellar ataxia* (see under cerebellar diseases, Fig. 189).

4. *Multiple or disseminated sclerosis* (Fig. 172) occurs between the ages of 15 and 30. The cause is unknown.

Pathology. There are dozens or hundreds of scattered nodules of sclerosis distributed throughout the central nervous system, especially in the white matter. The myelin sheath is lost, but the axons appear to be spared; hence there are no tract degenerations, and conduction is impeded, not entirely abolished. The chief symptoms are due to defective conduction:

(*a*) Nystagmus.

(*b*) Slow indistinct scanning speech.

(*c*) Intention tremor, 4 to 8 per second, especially in upper extremities.

(*d*) Paraparesis.

(*e*) There may be bulbar, cerebral or cerebellar symptoms. The disease may come on slowly or rapidly and may last three months to twenty years with a tendency to exacerbations and remissions.

5. *Cross lesions of one lateral half of the spinal cord,* Brown-Sequard Paralysis (Figs. 109, 110, 112, 113 and 214). The cause may be traumatism, tumor, or a rare type of syringomyelia. The symptoms are all below the level of the lesion (Figs. 109 and 110).

Homolateral Symptoms

Nerve Tract Involved	*Symptoms*
Lateral cerebrospinal tract	Spastic paralysis below the lesion.
Lower motor neuron at the site of the lesion	Flaccid paralysis of segmental distribution for affected half segment.
Posterior column	Loss of muscle, tendon, joint sense, tuning-fork sense and compass sense below the lesion. Pain, heat and cold not lost.
Lowest sensory neuron	Complete anæsthesia of all forms of affected half segment.
Bulbospinal tract for vasomotor spinal centers	Vascular dilatation and possibly dryness of the skin.

Crossed Symptoms

Nerve Tract Involved	*Symptoms*
Spinothalamic tract after decussation of its constituents	Loss of pain, heat and cold on the opposite side of the body below the lesion.

For other symptoms see the text (page 255).

6. *Transverse interruption of the spinal cord, all tracts,* may be caused by softening, traumatism, tumor, or myelitis (see text and Fig. 217).

Lesions of the Cortex of the Hemispheres and of the Association and Commissural Fibers

Commoner lesions.

1. Tubercle, sarcoma, glioma, metastatic carcinoma, cyst, gumma, and other conditions of rarer occurrence. Bony spicules from the dura may be large enough to cause irritative symptoms.
2. Vascular lesions: Arteriosclerosis with narrowing, thrombosis and consequent softening or hemorrhage; embolism.
3. Traumatism.

Focal symptoms (Figs. 90 to 93, 138 to 147).

1. *Anterior central convolution* (motor area).

Irritative lesions cause localized convulsions (Jacksonian epilepsy), spreading all over the side supplied by the affected area first, then passing to the opposite side. The order of progression is always the same.

Destructive lesions cause monoplegia, with intermittent convulsions affecting members governed by neighboring foci.

There is always slight sensory loss in the affected limb, especially of sense of posture and weight.

2. *Posterior central convolution.* This is the chief primary center for sensory judgments (ordinary sensation).

Irritative lesions cause paræsthesia (numbness, tingling) and perhaps Jacksonian convulsions with a sensory aura.

A destructive lesion causes errors in tactile and postural judgments in a single limb, especially in the hand or foot, with possible irritative sympoms preceded by a sensory aura.

There may be ataxia of the affected limb.

A subcortical lesion of these two areas causes similar symptoms of somewhat wider distribution. In left-sided lesions in right-handed people there may be left handed apraxia owing to interruption of callosal fibers (Fig. 173).

3. *Posterior third of inferior frontal gyrus* (motor speech center). (Figs. 144, 167, 105 and 12.)

A destructive lesion (on the left side in a right-handed patient and vice versa) causes Broca's motor aphasia (Fig. 167). Rarely a right-sided lesion causes motor aphasia in a right-handed patient. A subcortical lesion causes pure word dumbness.

There may be accompanying Jacksonian convulsions of the opposite face or arm.

4. *Posterior end of superior and middle frontal convolutions* (Fig. 142). These are the centers for higher motor memories; especially on the left side in a right-handed patient their destruction may cause apraxia of the right hand, without paralysis but with apraxia of the left hand. (See apraxia.)

In the posterior end of the *middle frontal gyrus* there is an area of irritation which causes conjugate deviation of the head and eyes to the opposite side; while destruction causes deviation of the head and eyes to the side of the lesion by unopposed action of the opposite center. Irritation here may also cause dilatation of both pupils with hippus of the opposite pupil.

5. Tumors cortical or subcortical of the anterior part of the frontal lobe of the left hemisphere of a right-handed patient may cause alterations in mental and moral state, but the symptoms are too indefinite for focal diagnosis till a neighboring motor area is involved.

6. Injury to the deep projection fibers to and from either or both frontal lobes are shown by Marie and Behagere to be accompanied by loss of the sense of orientation in space, loss of sense of direction and locality (*Revue Neurologique,* 1919, page 3).

7. *Anterior ends of supramarginal and superior parietal convolutions* (Fig. 142). Lesions cause contralateral tactile agnosia (astereognosis) and apraxia (ideational apraxia?).

8. A lesion of the left angular gyrus, the center for visual memories of words in a right-handed patient, causes inability to read—optic aphasia or alexia (Fig. 105, 9). If deep enough to injure the geniculo-thalamo-occipital (optic) radiation, there is homonymous hemianopsia (Fig. 105, g).

A subcortical lesion beneath the angular gyrus may cause pure word blindness.

A lesion here also causes difficulty in directing the eyes; there is paralytic

deviation of the eyes toward the lesion if it be destructive; or convulsive deviation from the lesion if it be irritative.

9. *Temporal lobe* (auditory centers).

On the right side of the brain in right-handed people this lobe is silent (Fig. 145); each ear has bilateral representation in the cortex.

On the left side in a right-handed patient a lesion of the posterior half of the superior temporal convolution causes Wernicke's sensory aphasia (Fig. 105, 11).

A subcortical lesion of the left side in the right-handed causes pure word deafness.

Bilateral destruction of the auditory area of the superior temporal gyrus causes cortical deafness.

An extensive lesion of the left middle temporal gyrus may cause mind deafness.

Such lesions, especially if subcortical, may involve loss of power of speaking and reading. As most people carry on their mental processes by subconsciously talking to themselves, such lesions may be accompanied by progressive mental deterioration.

An irritative lesion here may cause Jacksonian convulsions preceded by an auditory aura.

10. *Gyrus Hippocampi, pyriform area* (center for smell).

An irritative lesion here may cause Jacksonian fits, smacking of the lips, and tongue movements preceded by an aura of smell and taste, accompanied by a curious dreamy state with visual hallucinations (uncinate fits), with possibly later more generalized convulsions.

11. *Insula.* As this area on the left side lies between the auditory, motor speech, and word seeing centers, lesions here, especially those involving the white matter (association fibers) cause complex aphasias.

12. *Calcarine area* (Figs. 105 to 108). The area striata surrounding the calcarine fissure is the primary cortical visual center for the homonymous half of each retina. A lesion here causes blindness of the opposite field of vision for both eyes; that is, a lesion of the left calcarine area causes right homonymous hemianopsia, and vice versa.

A lesion of the white matter of the occipital lobe which interrupts the optic radiation causes the same symptoms. If on the left side in right-handed people, it interrupts in addition association fibers from the calcarine area to the angular gyrus, it causes word blindness.

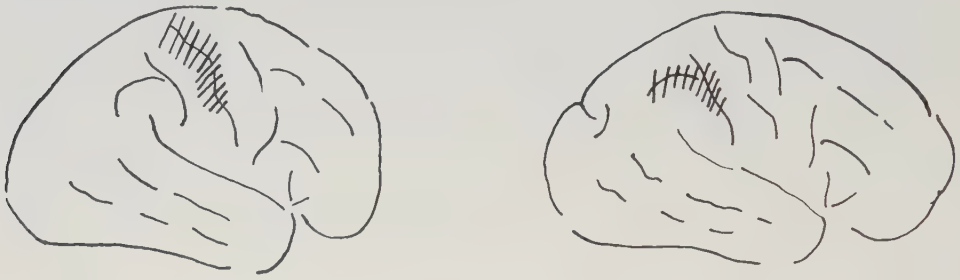
A lesion of the association fibers alone on the left side in right-handed people which spares the optic radiation may cause word blindness or loss of other forms of sight memories (mind blindness) without blindness to visual images; that is, the patient may not be able to interpret what he sees in terms of previous experiences.

Bilateral destruction of the calcarine area causes cortical blindness without loss of light reflexes; central vision is usually spared in cortical blindness (Fig. 105, 7 and 13).

13. *Corpus callosum.* A lesion of the genu and anterior part of the corpus callosum causes left-handed apraxia without paralysis (in right-handed patient). Fig. 173.

Silent Areas. All cortical and subcortical areas not mentioned above may be extensively diseased without localizing symptoms. They are called silent areas and are much more extensive in the right than in the left hemisphere (in right-handed people). (See figures of cerebral localization, 143, 144 and 145.)

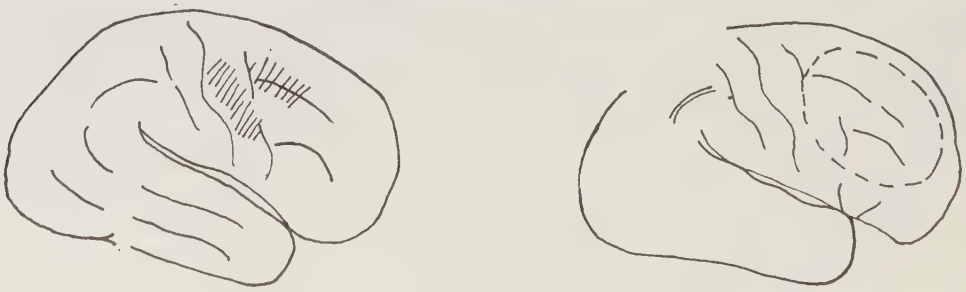
The chief site of silent tumors after the right temporal lobe is the right frontal lobe. Dreamy states are characteristic of silent tumors of the temporal lobes, most frequently the right. (Consult Cerebral Localization, Oxford Loose Leaf Medicine.)



F G

Fig. F. Site of tumor causing spasm beginning in left thigh extending to body, arm, and face. (Starr.)

Fig. G. Tumor causing spasm of left fingers and thumb, then wrist, elbow and shoulder. Sensory phenomena not mentioned. Should have been sensory aura of tingling in fingers of left hand with atopognosis and astereognosis and defective sense of position in left arm or fingers.



H I

Fig. H. Area removed by Horsley for persistent athetosis of left fingers. Resulting paralysis of left arm with partial recovery later. Persistent loss of sense of position; some loss of tactile sense. See page 131.

Fig. I. Endothelioma of right hemisphere. Symptoms for 2 years; no mental symptoms except perhaps a little emotionalism, ascribed to hysteria. Paresthesia of left hand; then paresis successively of hand, fingers and arm. After 4 months left leg dragged, arm hung by side in slightly flexed position. Later, pain became rather prominent, leading to diagnosis of thalamic thrombosis. Reflexes diminished. Choked disc did not appear till one year after first symptoms. One year after first symptoms tremor of *right* fingers and forearm; paresis of left face. Right pupil larger than left; Corneal reflex absent on right side. Vomiting and headache appeared late. No mental clouding till 2 months before death. P.M.; Endothelioma 3″ x 2¾″ x 2⅛″ displacing internal capsule and flattening thalamus. Note absence of mental symptoms in right frontal tumor.

Fig. J. Site of large subcortical tumor. Sensory fits; aura commencing with sensation of pins and needles in third or fourth fingers of left hand, spreading to arm, leg, trunk, and face of same side, followed some weeks later by motor spasms starting in left side of mouth and spreading to arm and leg of same side. Operation: recovery with some tactile atopognosis.

J

Fig. K. Position of cyst causing spasm of right shoulder extending down arm to fingers.

Fig. L. Situation of meningeal thickening and adhesions due to trauma, causing spasm of right leg beginning in extensors of knee and extending to foot and hip.

K

L

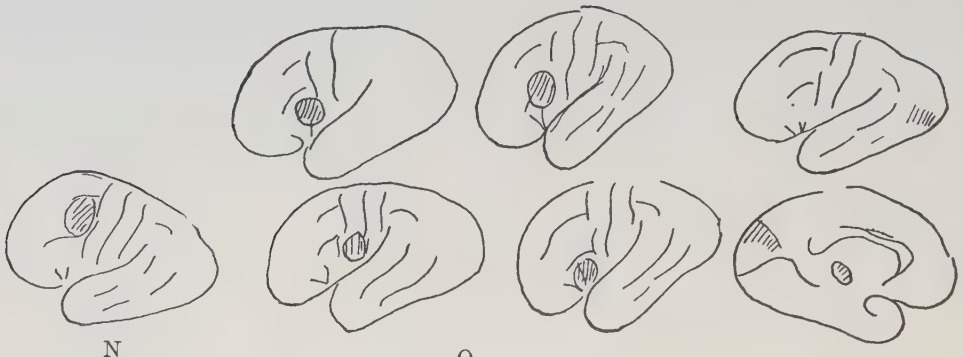
M

Fig. M. Site of tumor which caused spasmodic turning of head and eyes to the right, then forehead and cheek drawn to right (spasm of right facial nerve), motor aphasia.

Fig. N. Lesion causing spasmodic conjugate deviation of head and eyes to right.

Fig. O. Sites of lesions causing motor aphasia.

Fig. P. Site of melanotic sarcoma causing flashes of light referred to right eye (left half of both retinæ) with paroxysmal headache and



N O P

temporary complete blindness. There was also a flushing of the face during paroxysms.

Well marked optic neuritis. No hemianopsia. Mental condition clear. No paralysis. (Bramwell.)

Fig. Q. Site of lesions causing word deafness (sensory aphasia).

Fig. R. Site of lesions causing word blindness (alexia).

Q R

TABLES OF SEGMENTAL SUPPLY OF MUSCLES
MODIFIED FROM PURVES STEWART

TABLE 1
CERVICAL ENLARGEMENT

<table>
<tr><th>C1</th><th>C2</th><th>C3</th><th>C4</th><th>C5</th><th>C6</th><th>C7</th><th>C8</th><th>Th1</th><th></th></tr>
<tr><td colspan="9">Hyoid depressors
Genio - hyoid
T r a p e z i u s
Lev. ang. scap.
D i a p h r a g m
(Starr - 2, 3, 4.)
Scaleni</td><td>NECK</td></tr>
<tr><td colspan="5">D e l - t o i d
Rhomboids
Serratus ant.
Pect. maj. - clav.
Supraspin.
(Starr - 4, 5.)
Subscapularis
Teres minor</td><td colspan="2">Pect. maj.
(sternal)
Latt. dorsi
Pect. min.</td><td colspan="2"></td><td>SHOULDER</td></tr>
<tr><td colspan="5">D e l - t o i d
(Starr - 4, 5.)
Biceps brachii
Brachialis ant.
(Starr - 6, 7.)
Brachio - radialis
(Starr - 4, 5, 6.)</td><td colspan="3">T r i c e p s</td><td colspan="2"></td><td>ARM</td></tr>
<tr><td colspan="5"></td><td>Flex. & Ext. of Wrist
Pronators</td><td>Flex. & Ext. of Fingers</td><td colspan="2"></td><td>FOREARM</td></tr>
<tr><td colspan="5">Starr:
Long extensors of wrist, 6, 7.
Long extensors of fingers, 7.
Long flexors of wrist and fingers, 7, 8.
Extensors of thumb, C8, Th.1.</td><td></td><td></td><td colspan="2">Interossei
Lumbricals
Short mus. of hand</td><td>HAND</td></tr>
</table>

TABLE 2

LUMBO-SACRAL CORD

Th12	L1	L2	L3	L4	L5	S1	S2	S3	S4	
Quadratus lumb Cremaster P s o a s Iliacus (Starr - 1, 2.)					Tensor fasc. fem. Gluteus medius Gluteus minimus Quadratus femoris Gemelli Gluteus maximus Obturator internus					HIP
		Pectineus Adductor long. G r a c i l i s Adductor brevis Sartorius (Starr - 2.) Obturator exter. Quadriceps extens. (Starr - 2, 3.)		Adductor magnus (Starr - 3, 4.)	S e m i m e m b r a n o s u s (Starr - L5, S1.) Semitendinosus (Starr - 5.) Biceps femoris (Starr - L5, S1.)					THIGH
				Tibialis anterior (Starr - S2.) Extens. prop. hal. Extensor long. digitorum	G a s t r o c n e m i u s S o l e u s Plantaris Popliteus Peronei (Starr - 2, 3.) Tibial. post. (Starr - 1, 2.) Flex. long. dig. Flex. long. hal.					LEG
					Short muscles of the foot (Starr - S2, 3.)					Foot
						Sphincter ani (Starr - 4, 5.)	Levator ani Perineal muscles			

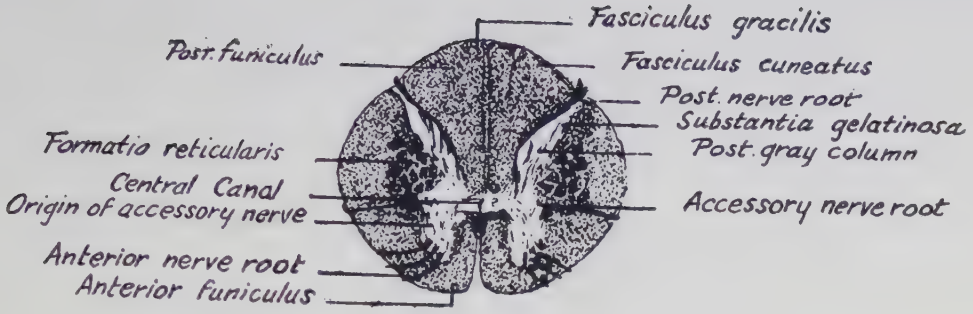


FIG. 1.—Section through adult spinal cord about the level of second cervical nerve. Weigert-Pal staining. (After Cunningham.)

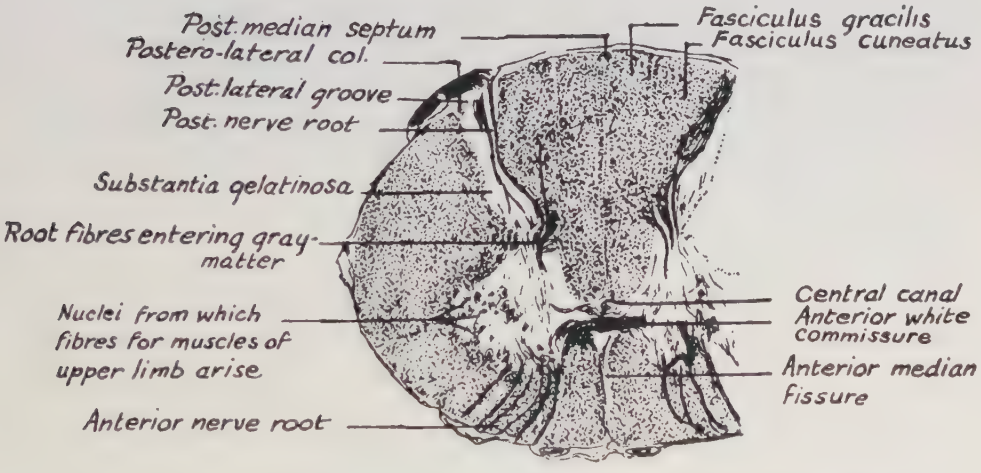


FIG. 2.—Section of adult spinal cord at fifth cervical segment. Weigert-Pal staining. (After Cunningham.)

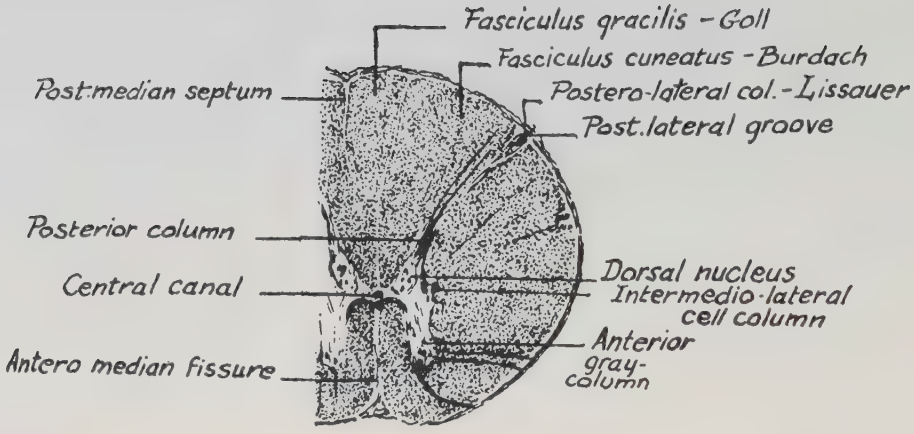


FIG. 3.—Section of adult spinal cord in mid-thoracic region. Weigert-Pal staining. (After Cunningham.)

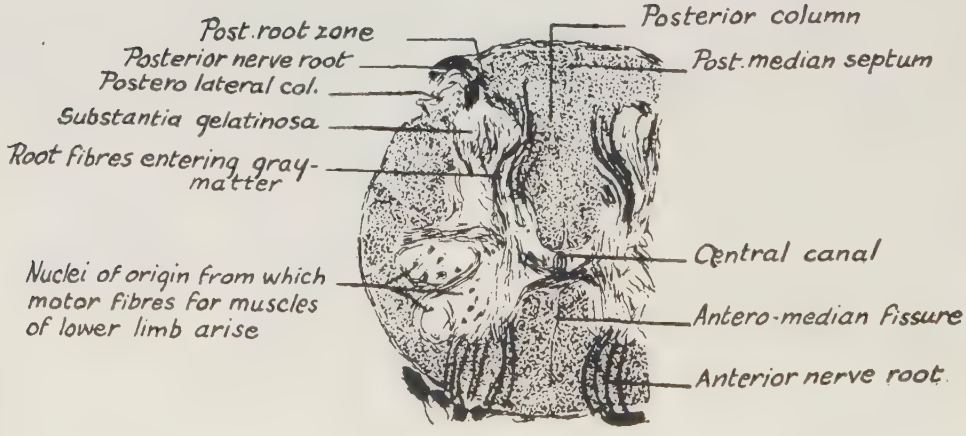


FIG. 4.—Section of adult spinal cord through fourth lumbar segment. Weigert-Pal staining. (After Cunningham.)

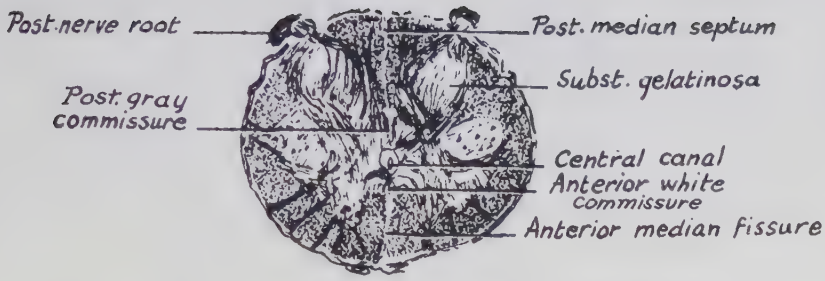


FIG. 5.—Section of adult spinal cord through third sacral segment. Weigert-Pal staining. (After Cunningham.)

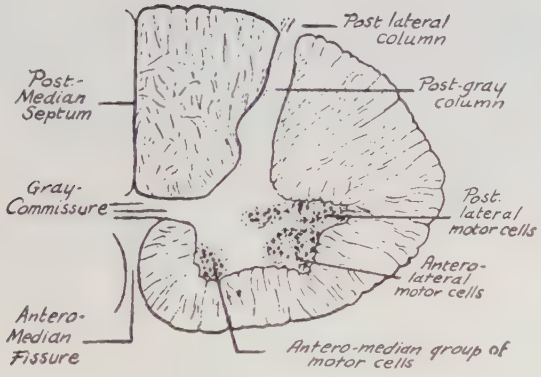


FIG. 6.—Section through fifth cervical segment of spinal cord to show grouping of cells in anterior gray column. (After Cunningham.)

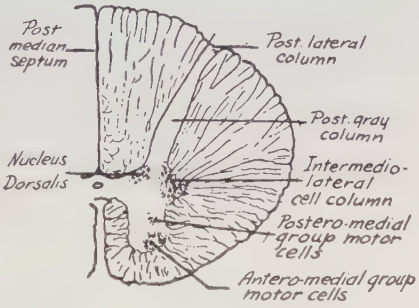


FIG. 7.—Section through eighth thoracic segment of spinal cord to show grouping of nerve cells. (After Cunningham.)

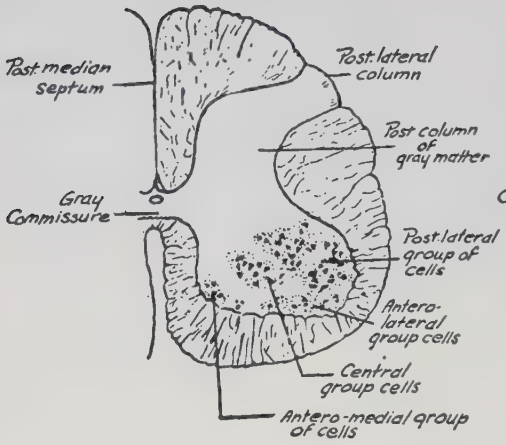


FIG. 8.—Section through the third lumbar segment of the spinal cord to show the grouping of the nerve cells of the anterior gray column. (After Cunningham.)

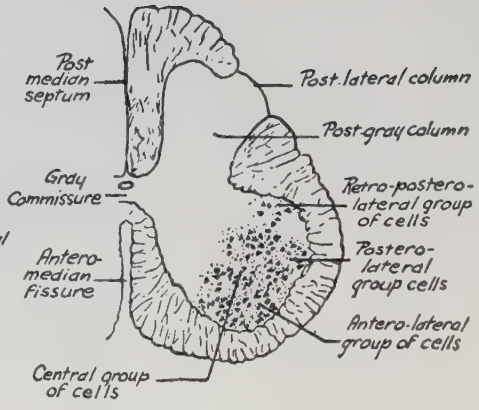


FIG. 9.—Section through the first sacral segment of the spinal cord to show the grouping of the nerve cells of the anterior gray column. (After Cunningham.)

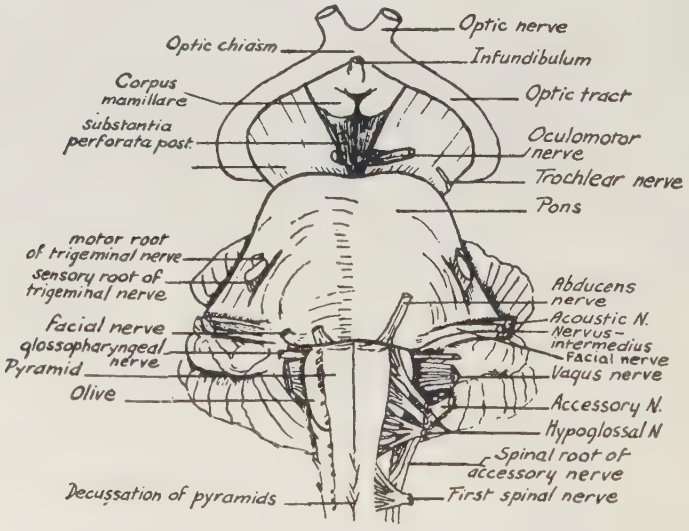


FIG. 10.—Ventral surface of the brain stem to show the superficial origins of the cranial nerves.

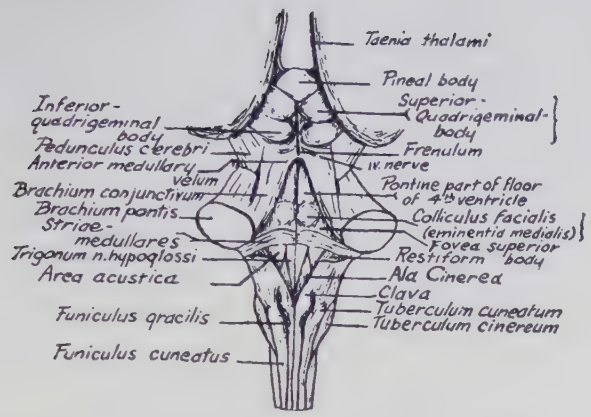


FIG. 11.—Dorsal view of the medulla oblongata, pons and mescencephalon of a full time fœtus. (After Cunningham.)

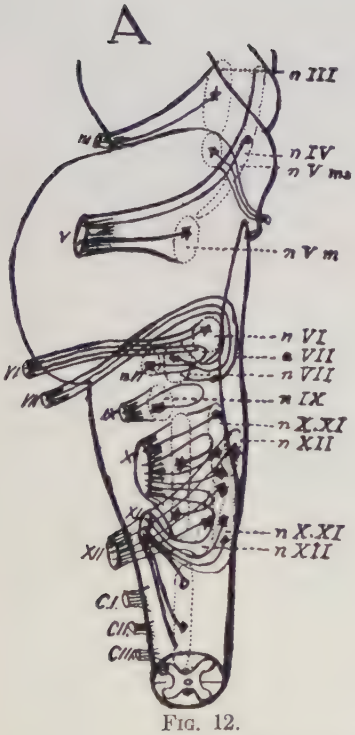


FIG. 12.

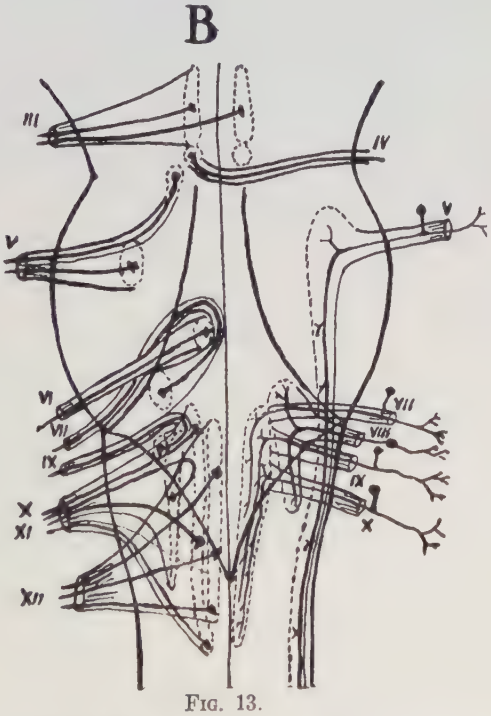


FIG. 13.

FIGS. 12 and 13.—Diagrams illustrating the origins and relations of the nuclei and root fibers of the cranial nerves. A. Efferent nerves only; profile view. B. On the left the motor nuclei and efferent nerves, except those of the IV nerve, and on the right side the afferent nerves and their terminal nuclei, surface view.

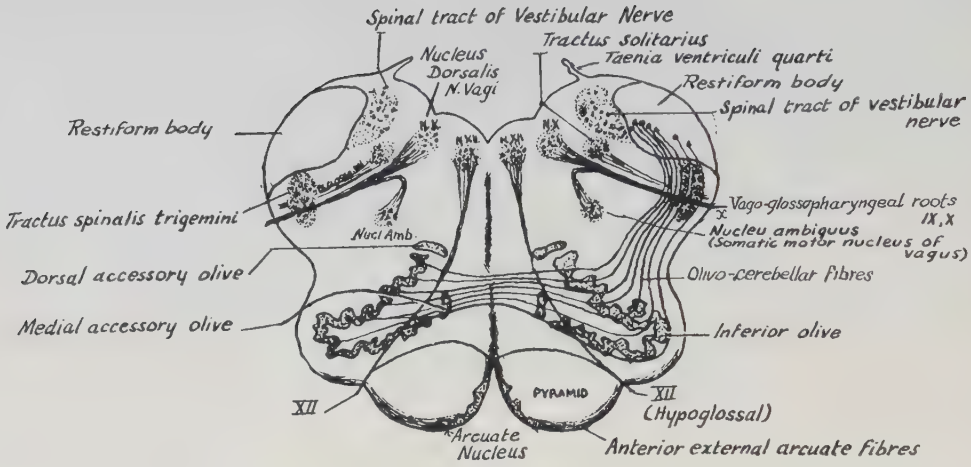


FIG. 14.—Diagram of the olivo-cerebellar fibers of the restiform body, also of the vago-glossopharyngeal and hypo-glossal nuclei and nerve roots. (After Cunningham.)

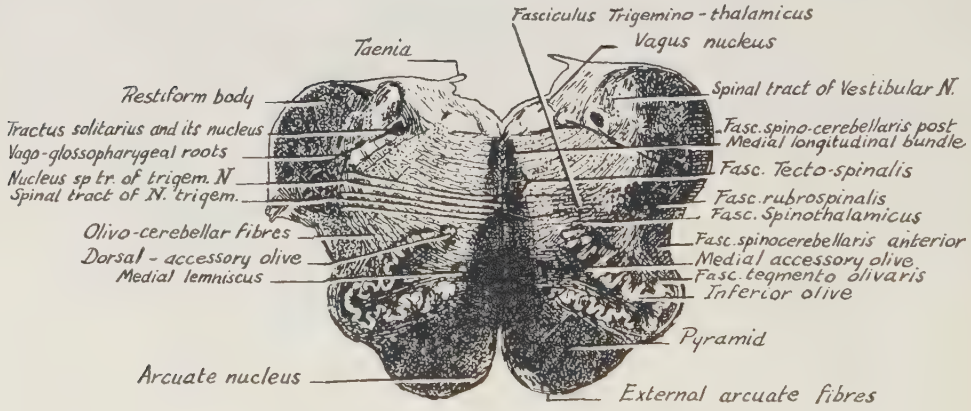


FIG. 15.—Transverse section through an adult medulla oblongata about the middle of the inferior olive. Weigert-Pal staining. (After Cunningham.)

Note: The fibers crossing from the nucleus of the spinal tract of the fifth nerve to the opposite trigemino-thalamic tract are diagrammatic.

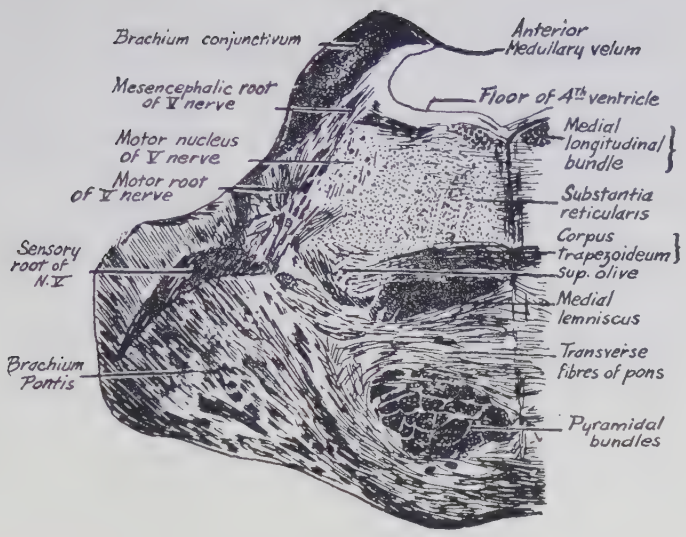


FIG. 16.—Transverse section through an adult pons at the level of the fiifth nerve. Weigert-Pal staining. (After Cunningham.)

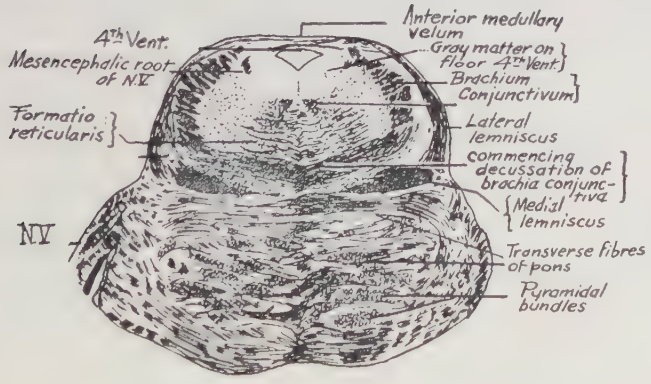


FIG. 17.—Transverse section through the upper part of an adult pons above the level of the trigeminal nuclei. Weigert-Pal staining. (After Cunningham.)

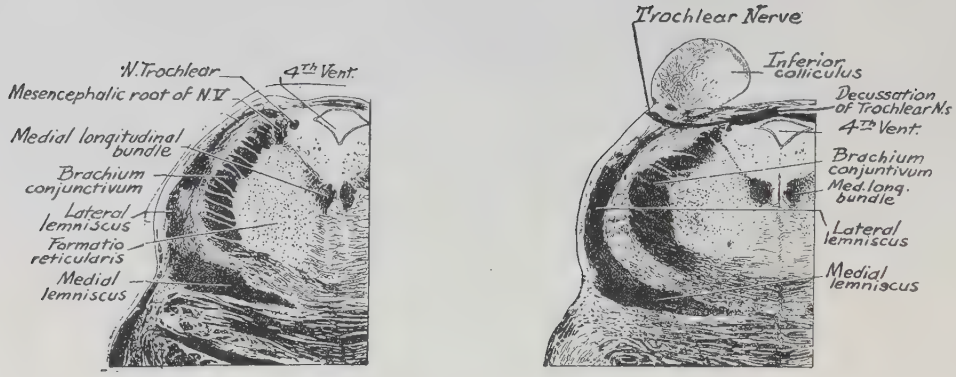


FIG. 18.—Transverse sections through the dorsal portion of adult pons at its superior part, close to the mesencephalon; right figure slightly higher than left. Weigert-Pal staining. (After Cunningham.)

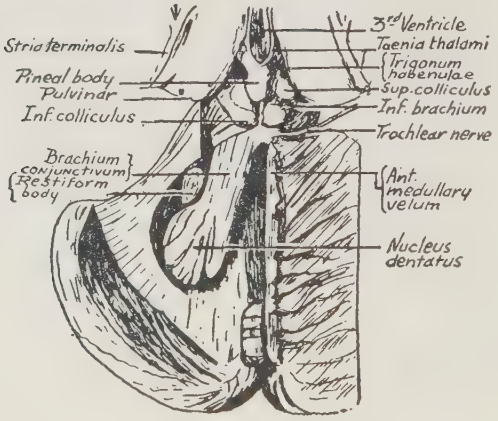


FIG. 19.—Dissection of the nucleus dentatus and brachium conjunctivum. (After Cunningham.)

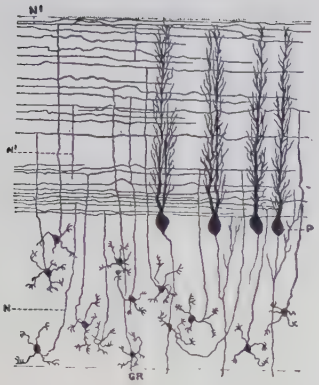


FIG. 20.—Section through the molecular and granular layers of a cerebellar folium in its long axis. Showing the cells and their processes as shown by the Golgi method of staining. P, cell of Purkinje; GR, granule cell; N, axone of granule cell; N^1, axone of granule cell in molecular layer. (Cunningham after Kölliker.)

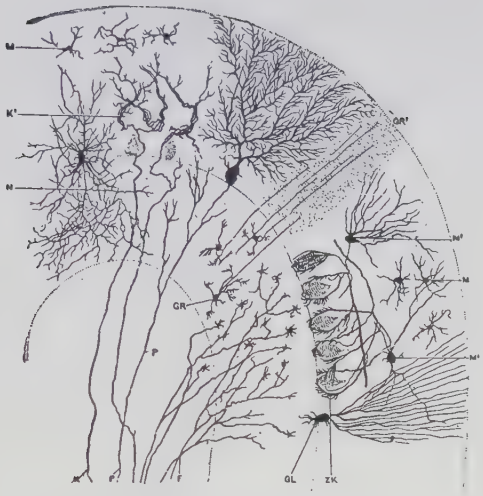


FIG. 21.—Transverse section through a cerebellar folium. Golgi stain. P, axone of cell of Purkinje; F, moss fibers; K and K^1, fibers from white substance of folium ending in the molecular layer in connection with the dendrites of the cells of Purkinje; M, small cell of molecular layer; GR, granule cell; GR^1, axones of granule cells in molecular layer; M^1, basket cells; ZK, basket work around cells of Purkinje; GL, neuroglia cell; N, axone of an association cell. (Cunningham after Kölliker.)

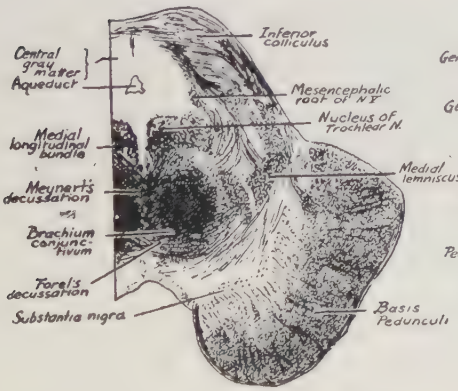


FIG. 22.—Transverse section through an adult mesencephalon at the level of the inferior colliculus.

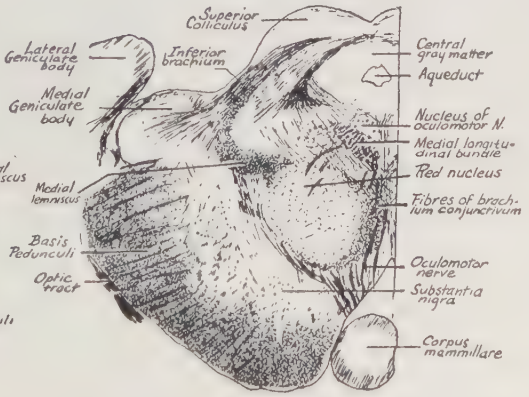


FIG. 23.—Transverse section through an adult mesencephalon at the level of the superior colliculus.

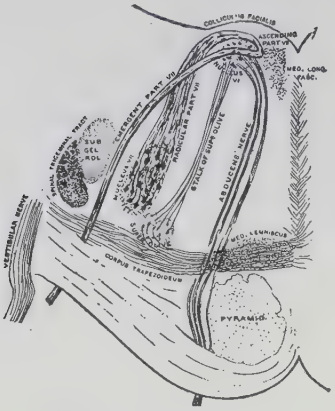


FIG. 24.—Diagram of the intrapontine course of the facial nerve and sixth nerve.

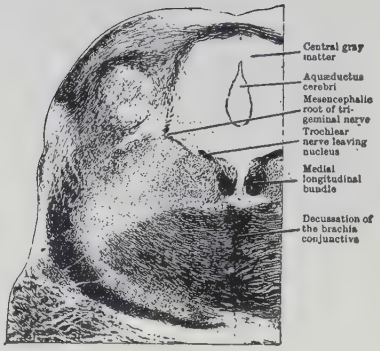


FIG. 25.—Section through the inferior colliculus and tegmentum of the mesencephalon at the level of the inferior part of the nucleus of the fourth nerve. (After Cunningham.)

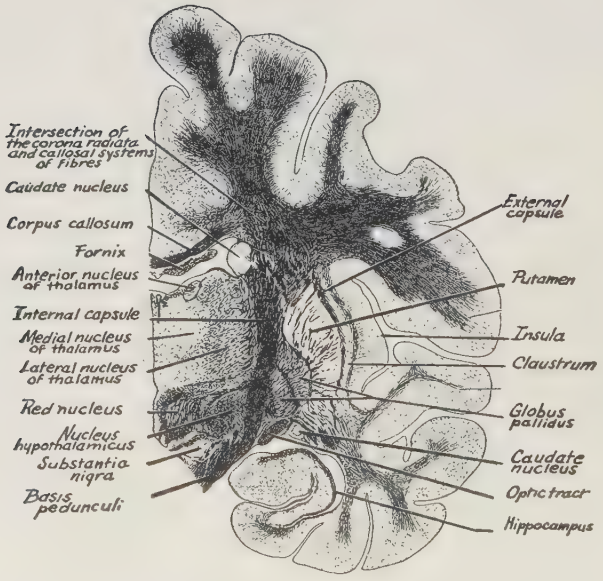


FIG. 26.—Frontal section through cerebrum of an orang in the hypothalamic tegmental region. Stained by the Weigert-Pal method. (After Cunningham.)

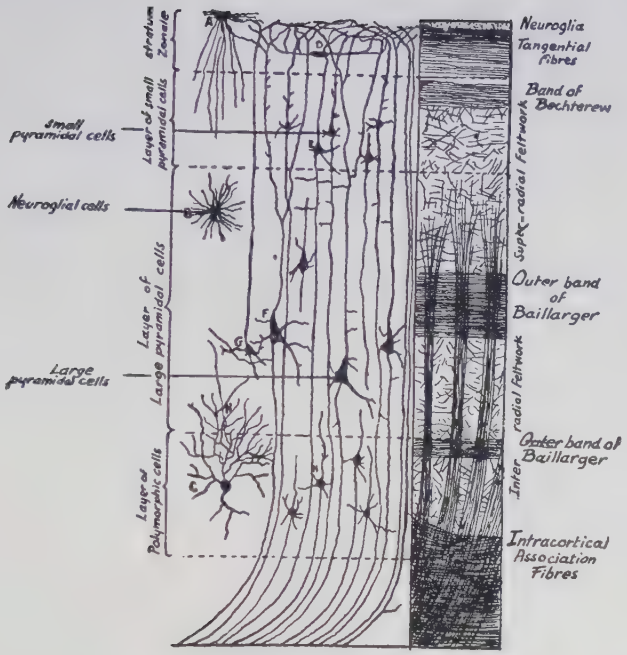


FIG. 27.—Diagram of minute structure of cerebral cortex. A, B, neuroglia cells; C, Golgi type II cell with short axone (N) which breaks up in a free arborization in the neighboring cortex; D, spindle-shaped cell in stratum zonale; E, small pyramidal cell; F, large pyramidal cell; G, cell of Martinotti; H, polymorphic cell; K, Corticicipetal fibers. (From Cunningham's Anatomy.)

FIG. 28.—The ventral aspect of prosencephalon (showing right optic tract). (From Cunningham's Anatomy.)

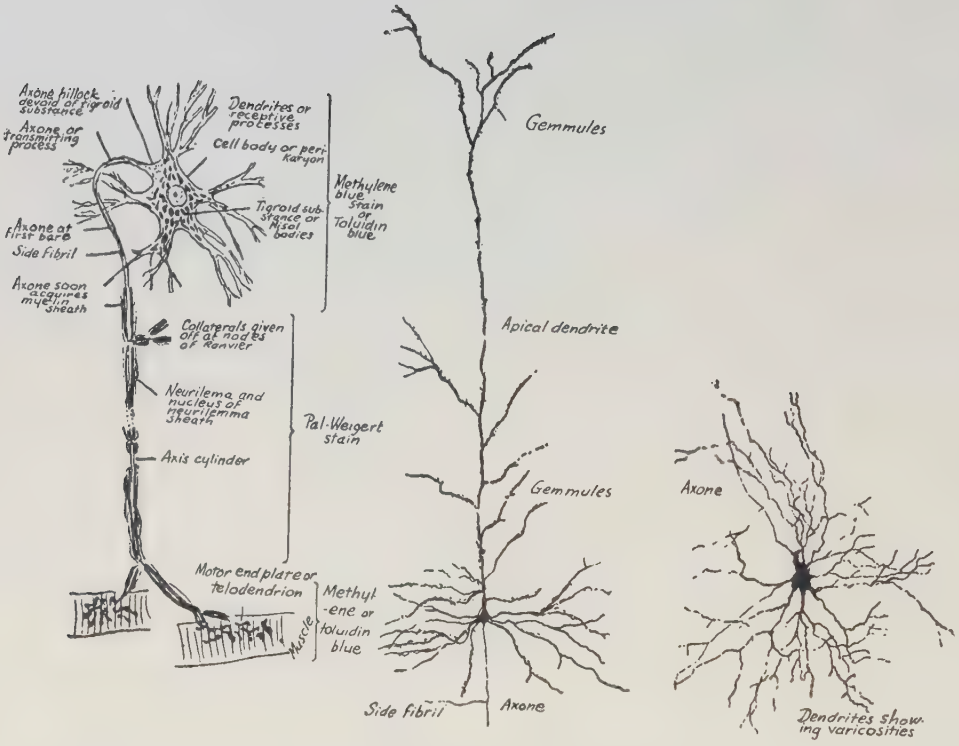


FIG. 29.—Composite figure of lower motor neurone to represent its constituents as brought out by different stains. The cell body, its immediate processes, and the end processes of the axone as stained by Nissl's technique with methylene blue or with toluidin blue, and the myelinated portion of the axone as stained by the Weigert-Pal stain or Heidenhain's iron hematoxylin stain for myelin. (After Barker.)

FIG. 30.—A pyramidal cell from the motor cortex of the cerebrum, as stained by Golgi's silver technique. A Golgi type I cell. (From Quain's Anatomy, after Cajal.)

FIG. 31.—A lower motor neuron cell from the anterior gray column of the spinal cord of a human fœtus; Golgi silver stain. A Golgi type I cell. (After Barker.)

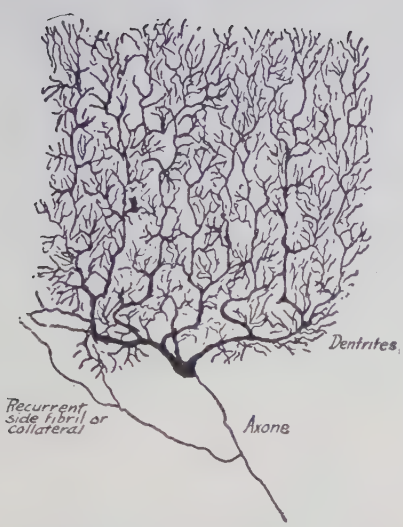


FIG. 32.—A Purkinje cell of the cerebellar cortex, Golgi stain. The axones leave the gray substance and travel some distance in the white center of the cerebellum. (From Quain's Anatomy.)

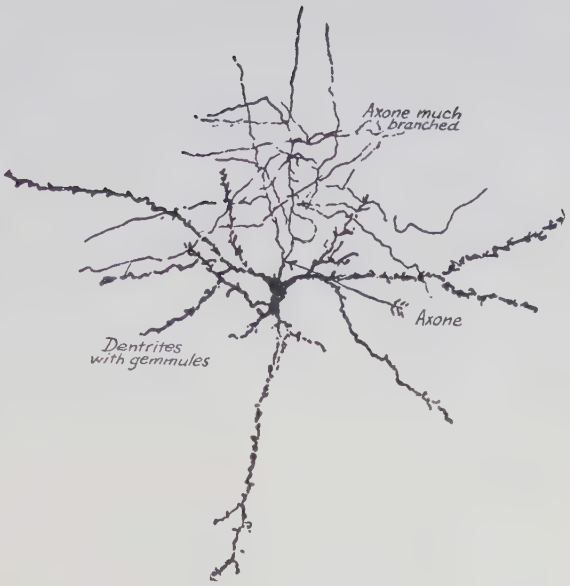


FIG. 33.—A Golgi type II cell from the cerebral cortex of a cat (compare Fig. 27c). Golgi silver stain. The axone branches freely and travels only a short distance in the neighboring gray matter. (After Barker.)

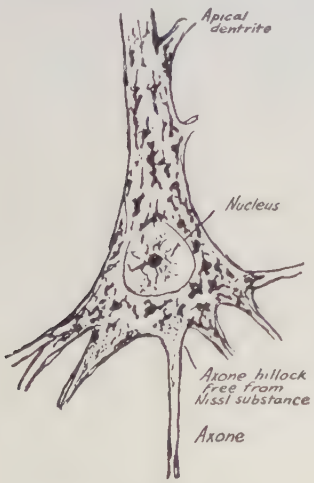


FIG. 34.

FIG. 35.

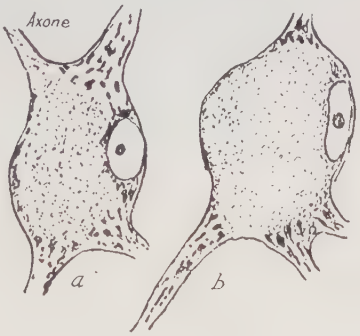


FIG. 36.

FIGS. 34, 35 and 36 are drawn to show Nissl bodies (tigroid substance) and chromatolysis resulting from fatigue or degeneration. Fig. 34—Large pyramidal cell of cerebral cortex. Nissl stain. Showing Nissl bodies (tigroid substance). (Cajal.) Fig. 35—Two motor cells from dog: (a) normal; (b) after prolonged fatigue. (Mann, Quain.) Fig. 36—Two motor cells from rabbit's spinal cord 15 days after section of the motor nerve fibers which arise from them. Nucleus displaced laterally. Chromatolysis (disappearance of tigroid substance). In (b) more advanced than in (a). (Cajal, Quain.) Stain: Nissl's methylene blue or toluidin blue. Special technique.

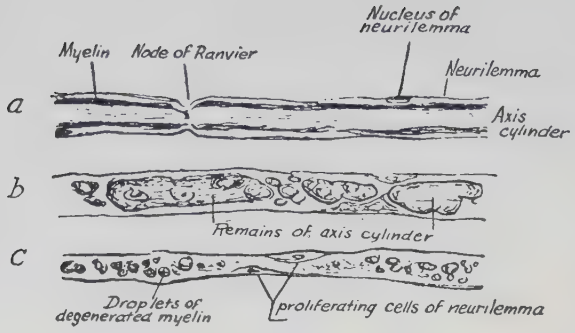


FIG. 37.

FIG. 38.

FIG. 39.

FIG. 40

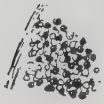


FIG. 41

FIG. 42

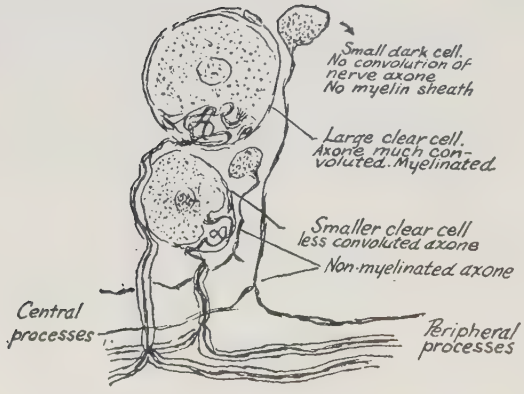

Small dark cell. No convolution of nerve axone No myelin sheath
Large clear cell. Axone much convoluted. Myelinated
Smaller clear cell less convoluted axone
Non-myelinated axone
Central processes
Peripheral processes

FIG. 43.

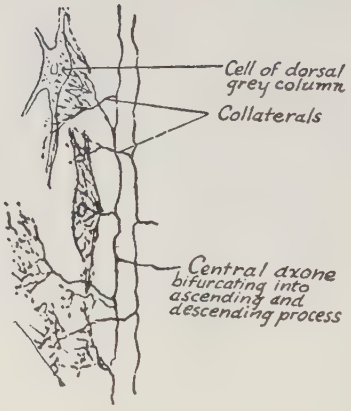


FIG. 44.

FIG. 37.—Wallerian degeneration of nerve fibers after section. Osmic acid stain: (a) normal nerve; (b) early stage; (c) more advanced. (Quain.)

FIG. 38.—Normal sciatic nerve of cat fixed (and stained) with osmic acid. The nerves vary in size. The myelin stains as distinct rings of black.

FIG. 39.—High power picture of portion of spinal cord stained by Marchi's method to show degenerated nerve fibers. Contrast with 38. When fixed in Mueller's fluid normal myelin does not stain with osmic acid, but degenerated myelin stains. Note irregular arrangement of the fat droplets as against the irregular myelin circles in normal nerve as seen in 38. Spencer lens Co 10x, Oc., 4 mm. Obj. = 400.

FIG. 40.—Low power picture of Fig. 39. 10x, Oc. 16 mm. Obj. = 100.

FIG. 41.—Degenerated nerve tract in glioma of cord. Scarlet red.

FIG. 42.—Cells from anterior horn of spinal cord. Same case as Fig. 39, showing fatty degeneration.

FIG. 43.—Types of cells from ganglion on posterior nerve roots of cat. Vital staining by methylene blue. (Cajal, Quain.)

FIG. 44.—Showing central processes of two sensory nerves in longitudinal section of spinal cord forming synapses with cells of posterior gray column: silver stain. (Cajal, Quain.)

Fig. 45.

Fig. 46.

Fig. 47.

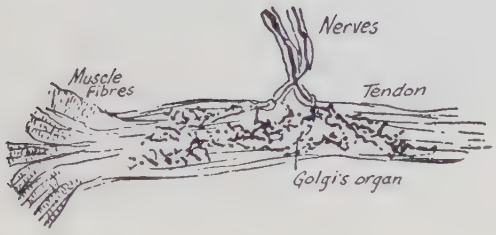


Fig. 48.

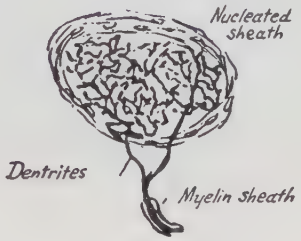


Fig. 49.

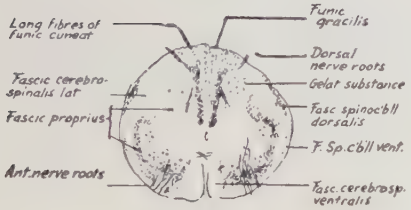


Fig. 50.

Fig. 51.

Fig. 45.—Plexus of sensory nerve endings in rabbit's cornea. Methylene blue. (Cajal, Quain.)

Fig. 46.—End bulbs in human conjunctiva. (Longworth, Quain.)

Fig. 47.—End bulb from human peritoneum. Methylene blue. (Dogiel, Quain.)

Fig. 48.—Sensory nerve ending in tendo calcaneus. (Ciaceis, Quain.)

Fig. 49.—End bulb from glans penis. (Dogiel, Quain.)

Fig. 50.—Section of cervical cord of 5½ mo. fœtus. Pal-Weigert stain. (After Alex Bruce.)

Fig. 51.—Section of oblongata of 5½ mo. fœtus. Pal-Weigert and Upson's carmine stain. (After Bruce.)

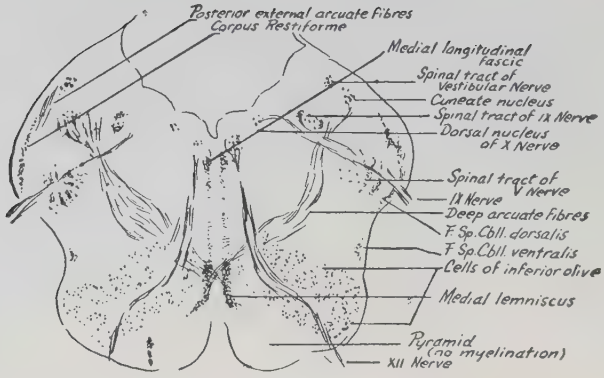


FIG. 52.—Section through middle of inferior olive of 5½ mo. fœtus. Pal-Weigert and Upson's carmine stain. (After Bruce.)

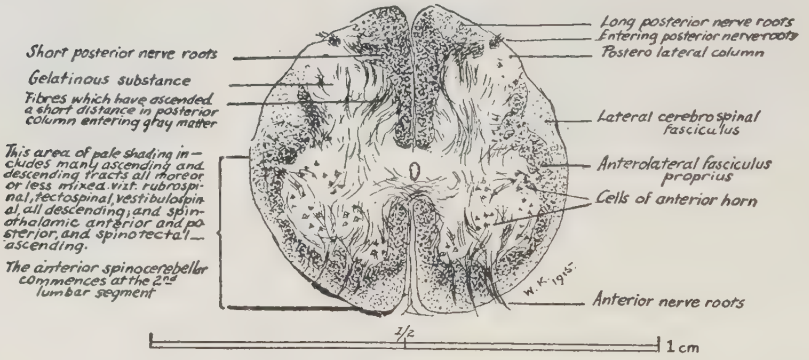

Short posterior nerve roots
Gelatinous substance
Fibres which have ascended a short distance in posterior column entering gray matter
This area of pale shading includes manly ascending and descending tracts all more or less mixed. viz: rubrospinal, tectospinal, vestibulospinal all descending; and spinothalamic anterior and posterior, and spinotectal—ascending.
The anterior spinocerebellar commences at the 2nd lumbar segment
Long posterior nerve roots
Entering posterior nerve roots
Postero lateral column
Lateral cerebrospinal fasciculus
Anterolateral fasciculus proprius
Cells of anterior horn
Anterior nerve roots
W. K. 1915.
1/2
1 cm

FIG. 53.—Section through lumbar enlargement of cord of full-time fœtus, stained for myelination. Pal-Weigert method. Cells of anterior gray column diagrammatic as regards size, but correct in distribution. Darker shading shows more advanced myelination. Drawn by aid of microprojection from specimen in Anatomical museum. Univ. of Texas.

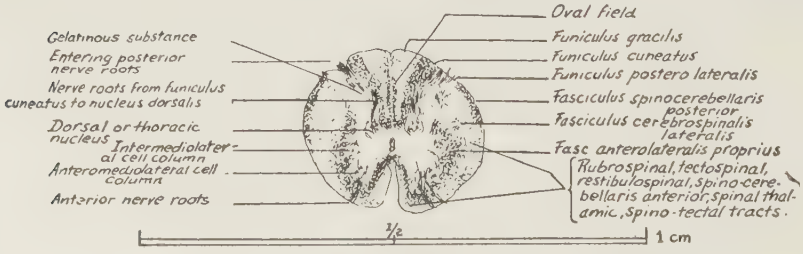


FIG. 54.—Section through 4th or 5th thoracic segment of fœtal cord; Pal-Weigert staining, size of cells exaggerated, rest drawn to scale. (W. Keiller.)

Funiculus gracilis
Long fibres of funic. cuneatus
Short fibres of funic. cuneatus
Spinal tract of V Nerve
Gelatinous substance
Formatio reticularis of cord (lateral cerebrospinal tract)
Emerging root of XI Nerve
Nucleus of XI Nerve
Anterolateral F. proprius
F. tectospinalis, vestibulospinalis, spinothalamicus anterior and posterior spinotectalis
Posterior root of 2nd cervical nerve
Post.r spinocerebellar fasciculus
Lateral cerebrospinal fascic. and fascic. rubrospinalis
Ant.r spinocerebellar fascic
Anterior roots of 2nd cervical
Fasc. cerebrospinalis anterior
1/2
1 cm

Fig. 55.—Cervical cord of fœtus at term. About 8th cervical segment. (W. Keiller.)

Fig. 56.—Fœtal cord about level of 2nd cervical nerve. (W. Keiller.)

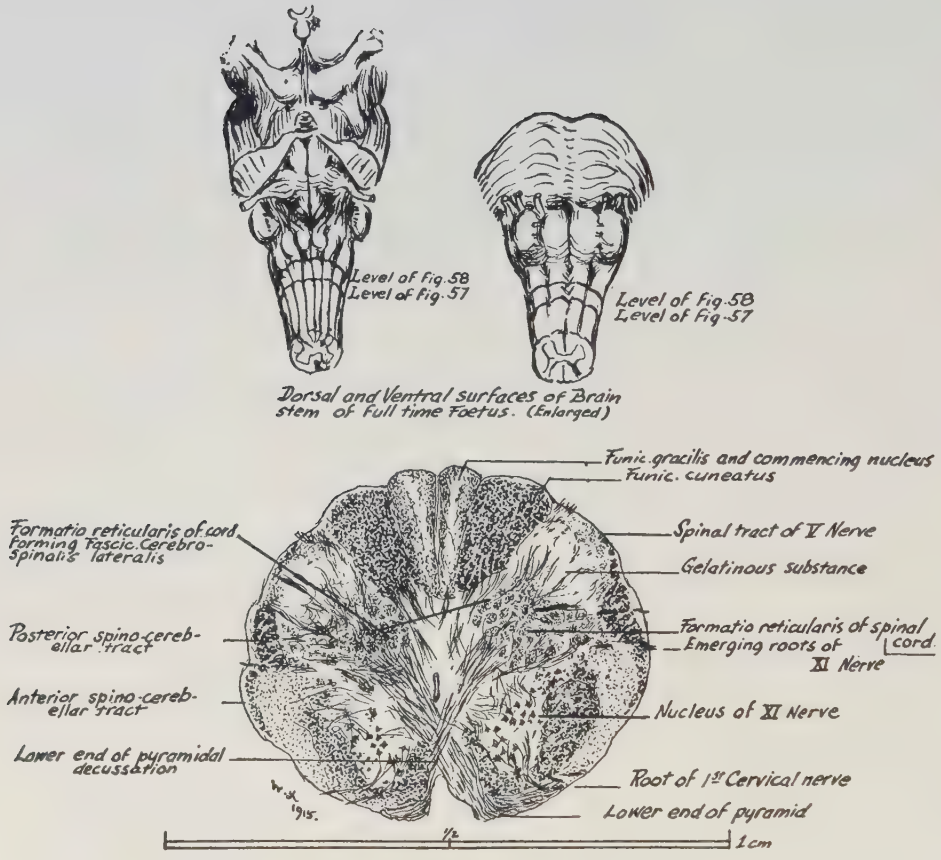

Level of Fig. 58
Level of Fig. 57
Level of Fig. 58
Level of Fig. 57
Dorsal and Ventral surfaces of Brain stem of full time Foetus. (Enlarged)
Funic. gracilis and commencing nucleus
Funic. cuneatus
Formatio reticularis of cord forming Fascic. Cerebro-Spinalis lateralis
Spinal tract of V Nerve
Gelatinous substance
Posterior spino-cerebellar tract
Formatio reticularis of spinal cord
Emerging roots of XI Nerve
Anterior spino-cerebellar tract
Nucleus of XI Nerve
Lower end of pyramidal decussation
Root of 1st Cervical nerve
Lower end of pyramid
W.K. 1915.
1/2
1 cm

FIG. 57.—Section of fœtal medulla oblongata at lower end of pyramidal decussation. (W. Keiller.)

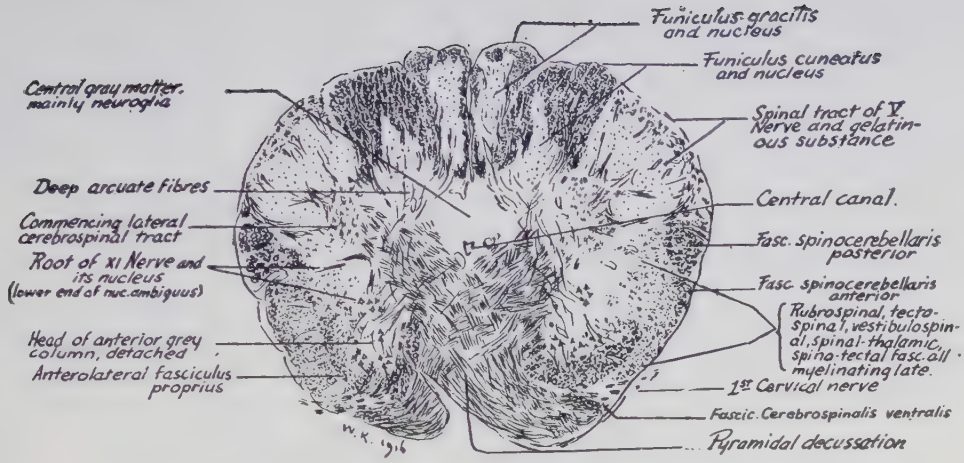

Funiculus gracilis and nucleus
Funiculus cuneatus and nucleus
Central gray matter, mainly neuroglia
Spinal tract of V. Nerve and gelatinous substance
Deep arcuate fibres
Central canal.
Commencing lateral cerebrospinal tract
Fasc. spinocerebellaris posterior
Root of XI Nerve and its nucleus (lower end of nuc. ambiguus)
Fasc. spinocerebellaris anterior
Rubrospinal, tectospinal, vestibulospinal, spinal-thalamic, spino-tectal fasc. all myelinating late.
Head of anterior grey column, detached
Anterolateral fasciculus proprius
1st Cervical nerve
Fascic. Cerebrospinalis ventralis
Pyramidal decussation

Fig. 58.—Section of fœtal medulla oblongata through middle of pyramidal decussation. (W. Keiller.)

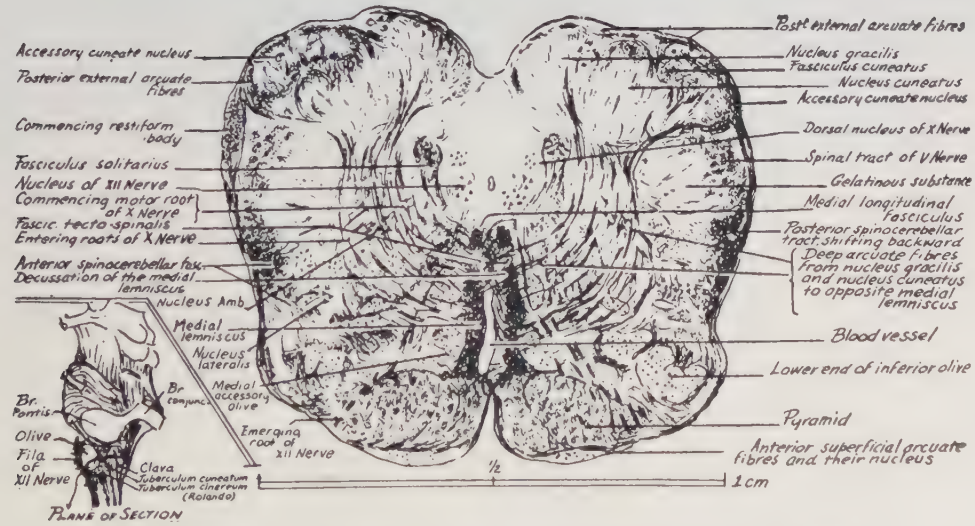


Fig. 59.—Section of oblongata of newborn child through lower end of inferior olive. (W. Keiller.)

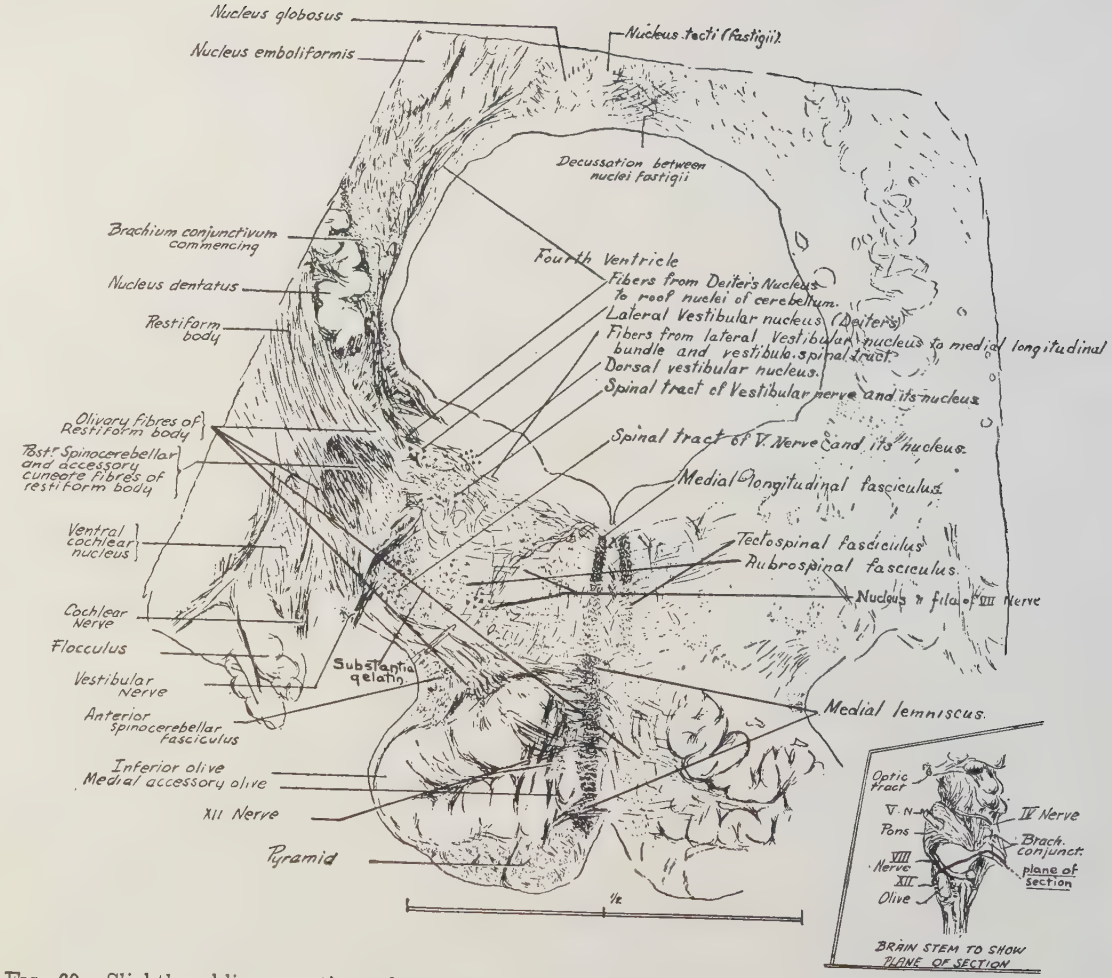


FIG. 60.—Slightly oblique section of upper end of oblongata of full time fœtus, for olivo-cerebellar fibers and Deiters' nucleus. Pal-Weigert hematoxylin. Only one-half of section shaded. (W. Keiller.)

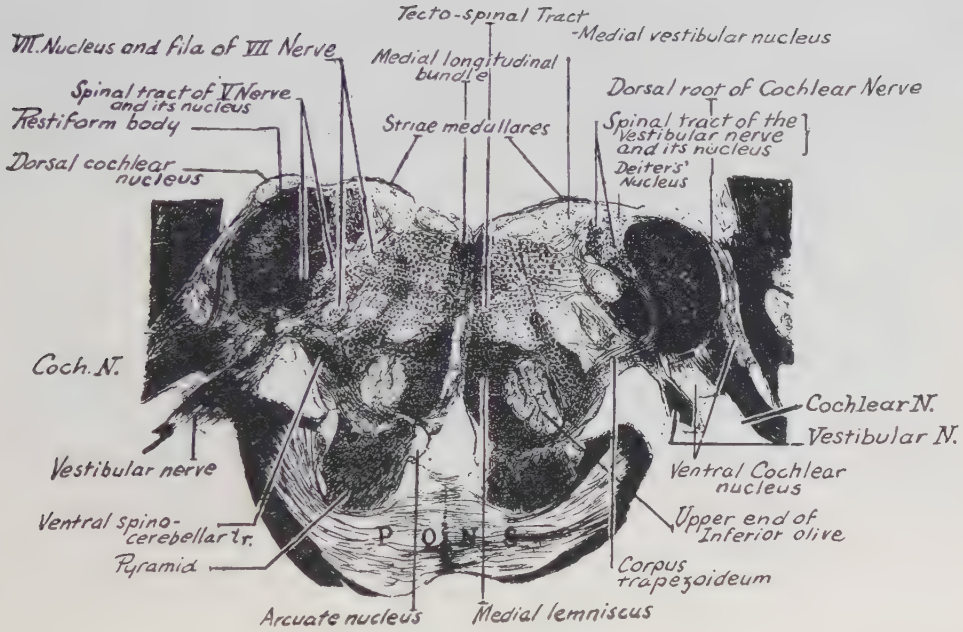


Fig. 61.—Section through junction of pons and medulla oblongata (adult). (W. Keiller.)

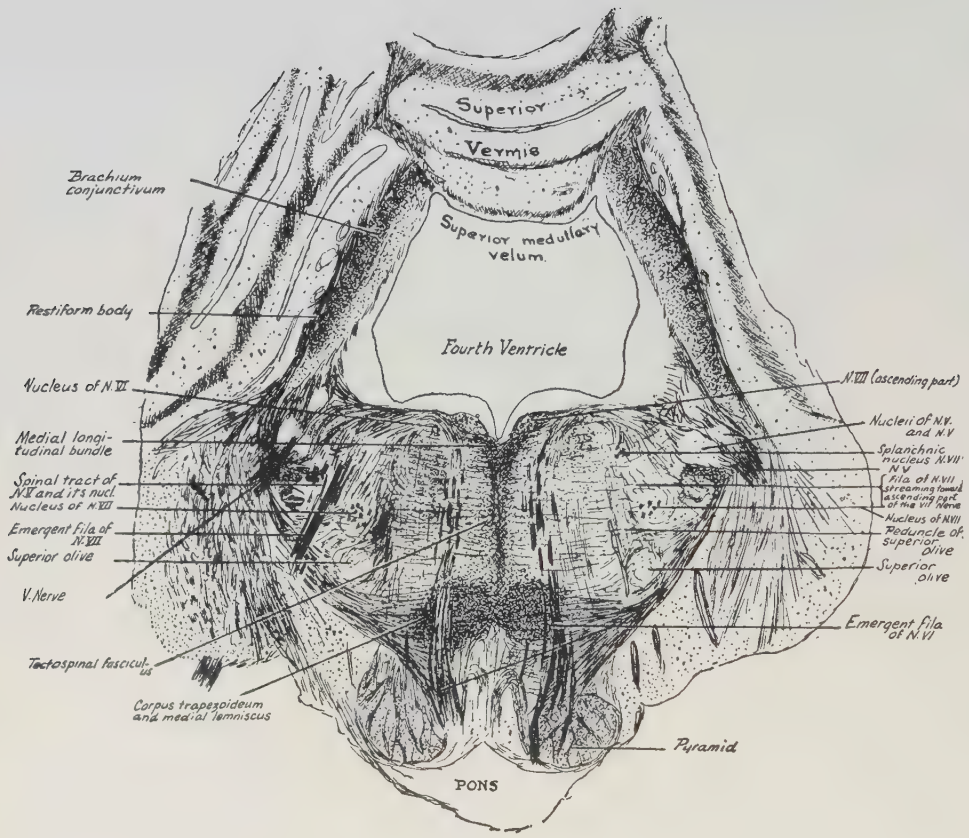


FIG. 62.—Section through brain stem of fœtus at junction of pons and medulla oblongata. (W. Keiller.)

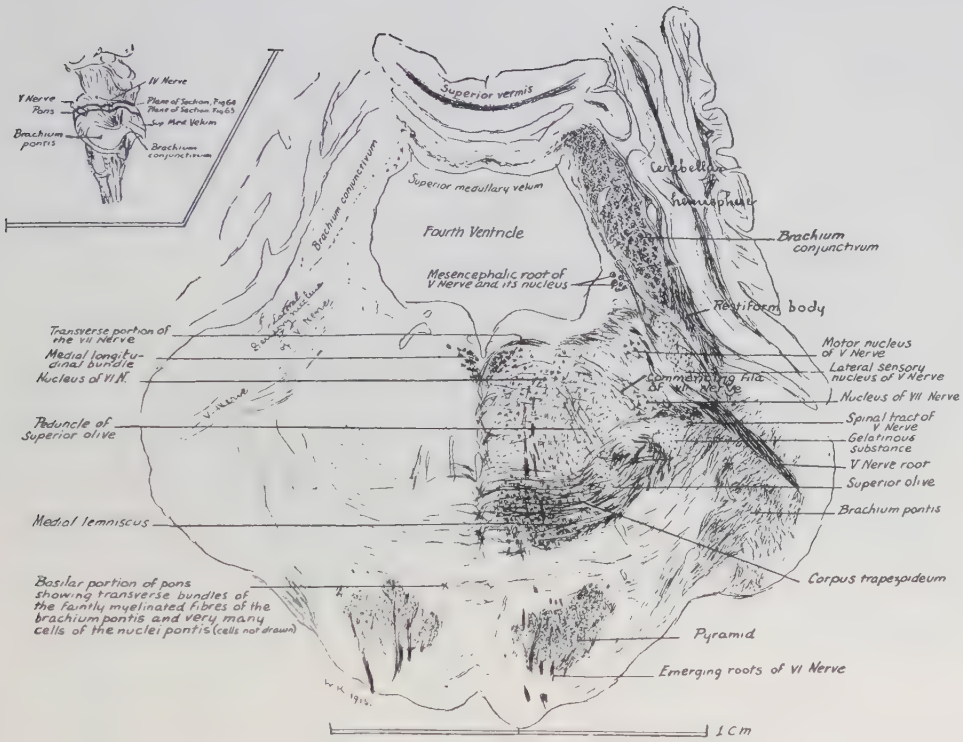


FIG. 63.—Section through middle of pons of full time fœtus. Shaded in on one side only. (W. Keiller.)

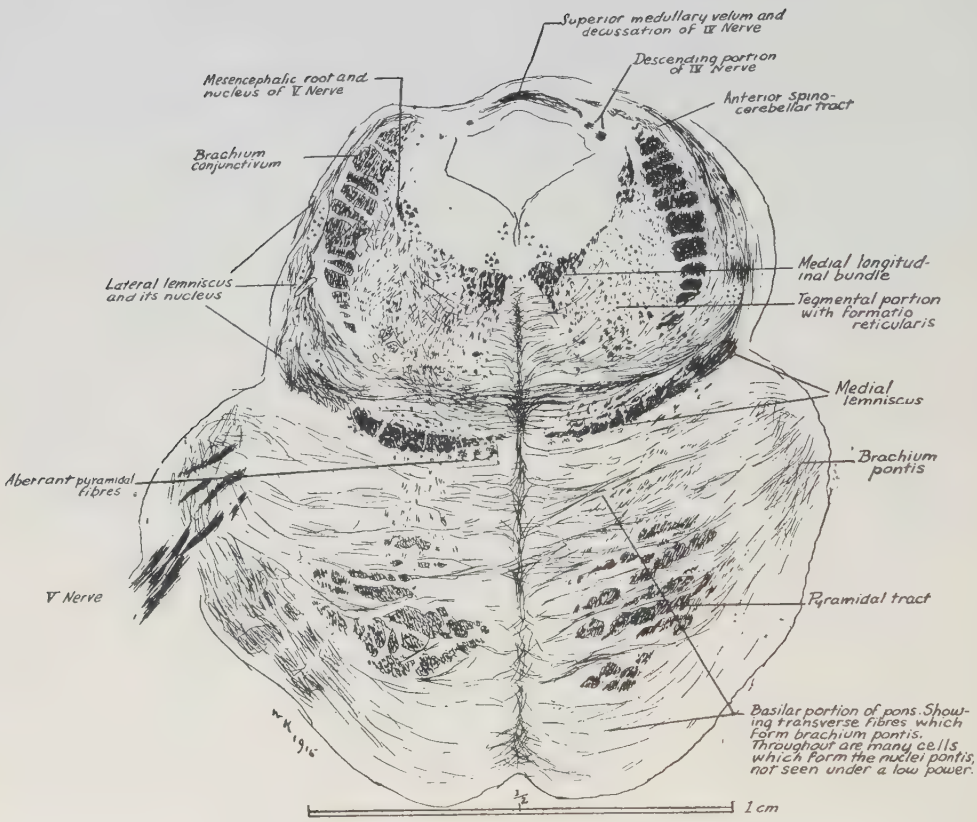

Superior medullary velum and decussation of IV Nerve
Descending portion of IV Nerve
Mesencephalic root and nucleus of V Nerve
Anterior spino-cerebellar tract
Brachium conjunctivum
Medial longitudinal bundle
Lateral lemniscus and its nucleus
Tegmental portion with formatio reticularis
Medial lemniscus
Brachium pontis
Aberrant pyramidal fibres
V Nerve
Pyramidal tract
Basilar portion of pons. Showing transverse fibres which form brachium pontis. Throughout are many cells which form the nuclei pontis, not seen under a low power.
W K 1916
1 cm

FIG. 64.—Oblique section through upper end of pons and superior medullary velum. Newborn child. Pal-Weigert stain. The section is rather higher on the right than on the left side. For plane of section see insert, Fig. 63. (W. Keiller.)

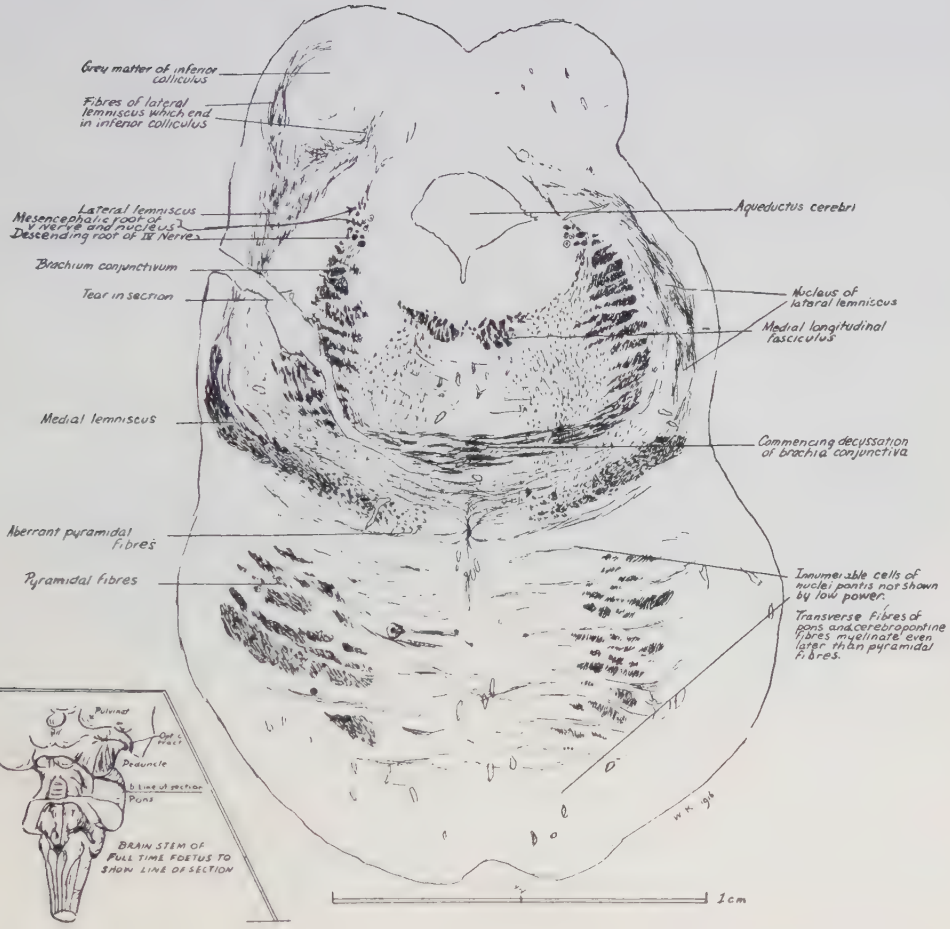

Grey matter of inferior colliculus
Fibres of lateral lemniscus which end in inferior colliculus
Lateral lemniscus
Mesencephalic root of V nerve and nucleus
Descending root of III Nerve
Brachium conjunctivum
Tear in section
Medial lemniscus
Aberrant pyramidal fibres
Pyramidal fibres
Aqueductus cerebri
Nucleus of lateral lemniscus
Medial longitudinal fasciculus
Commencing decussation of brachia conjunctiva
Innumerable cells of nuclei pontis not shown by low power.
Transverse fibres of pons and cerebropontine fibres myelinate even later than pyramidal fibres.
Pulvinar
Pons
BRAIN STEM OF FULL TIME FOETUS TO SHOW LINE OF SECTION
1 cm

FIG. 65.—Section through upper end of pons and inferior colliculus of full time fœtus. Pal-Weigert stain. Section rather higher on left side than on right.

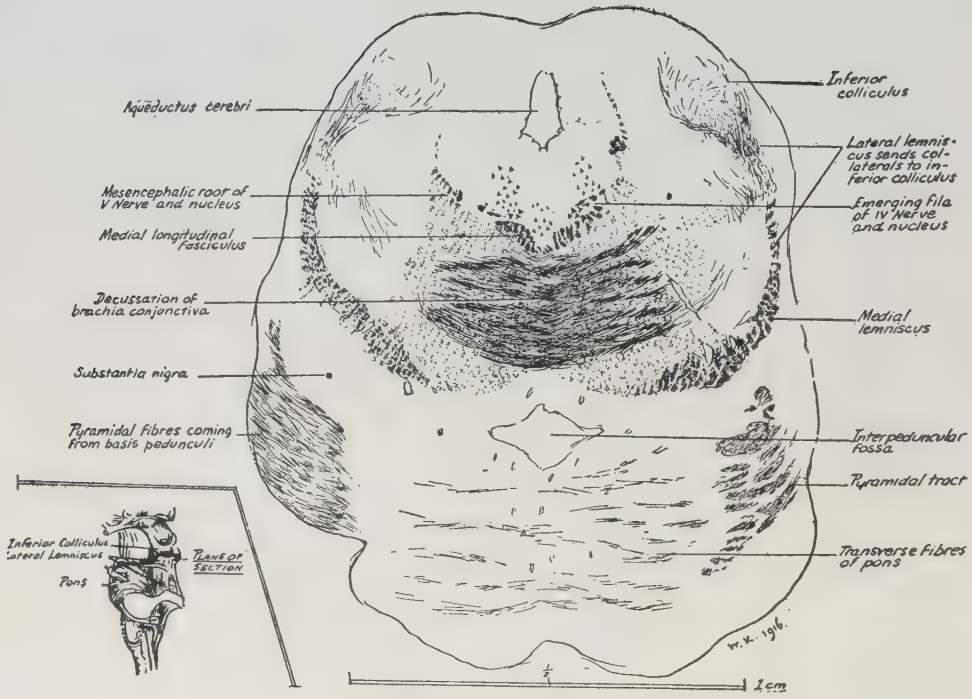


FIG. 66.—Transverse section through upper end of pons and inferior colliculi, a little higher on left side. Full time fœtus. Pal-Weigert stain. For plane of section see dark line round small figure. (W. K., 1915.)

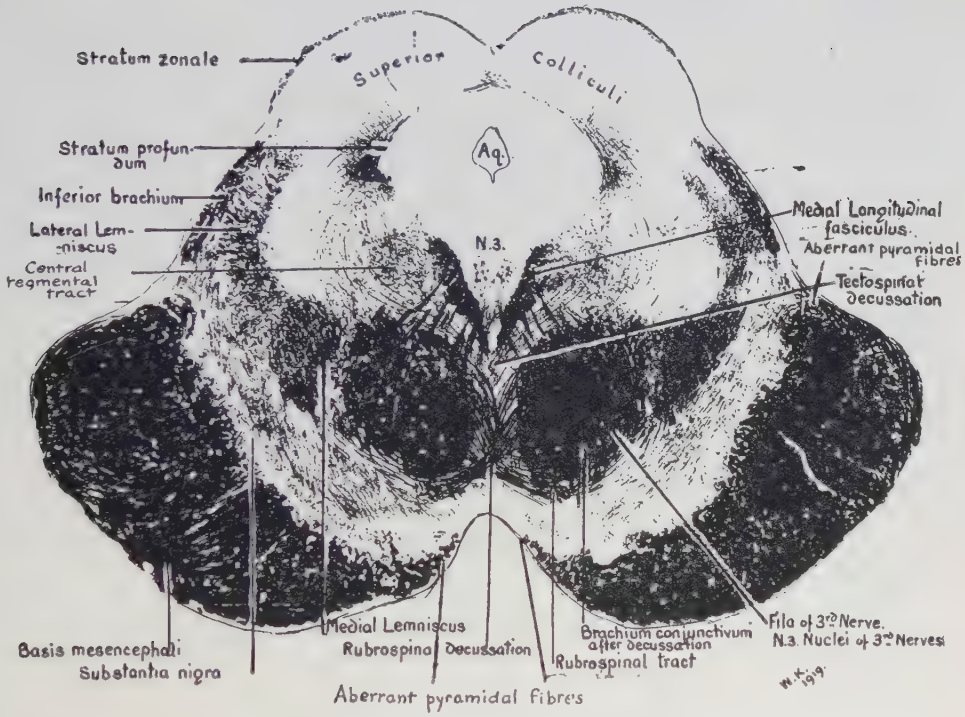


FIG. 67.—Section through superior colliculi to show tectospinal and rubrospinal decussations. Adult brain stem.

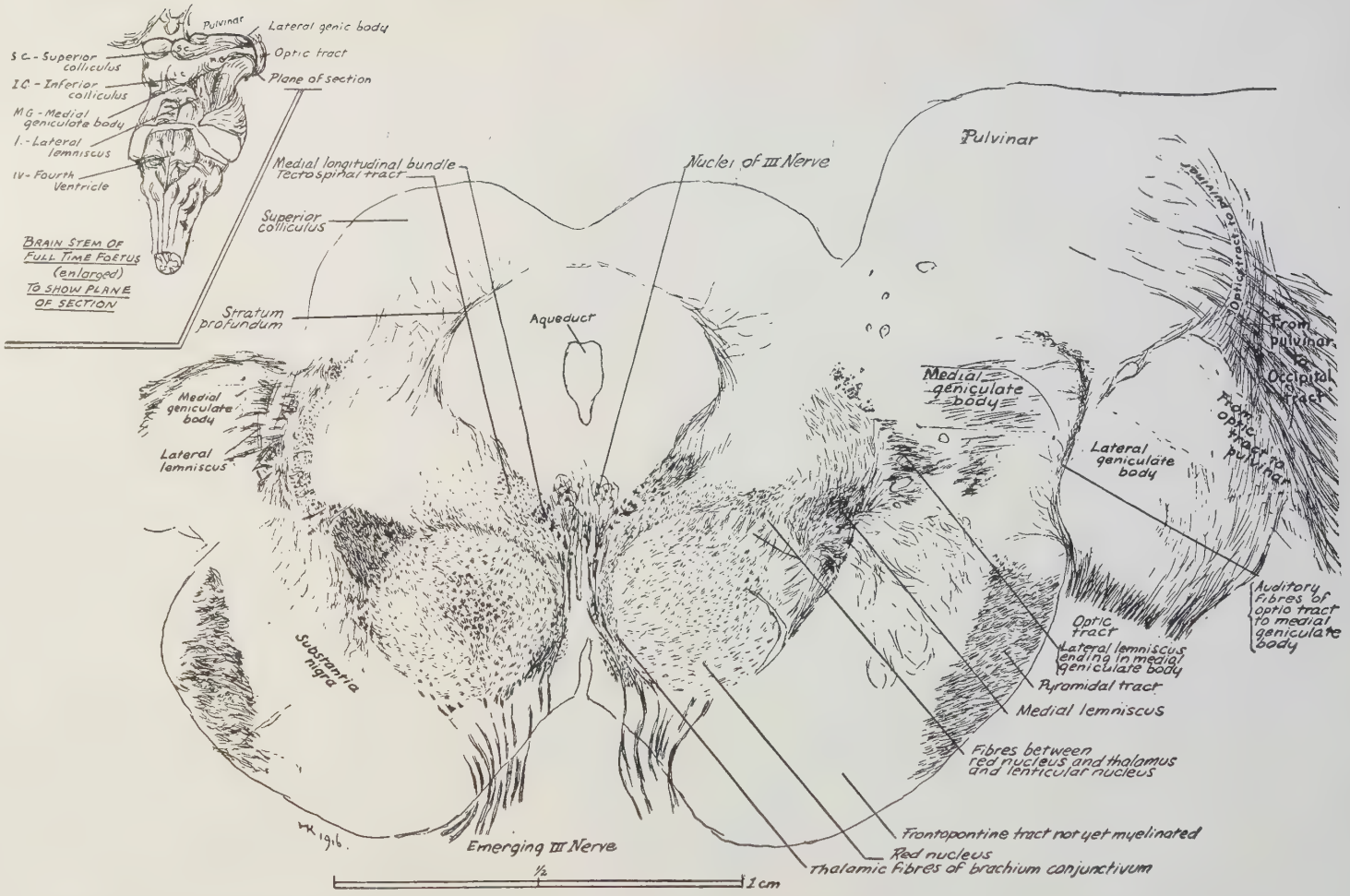


FIG. 68.—Through cerebral peduncles, superior colliculi and (on right side) pulvinar and optic tract. Full time fœtus. Pal-Weigert stain. Somewhat higher on right side than left.

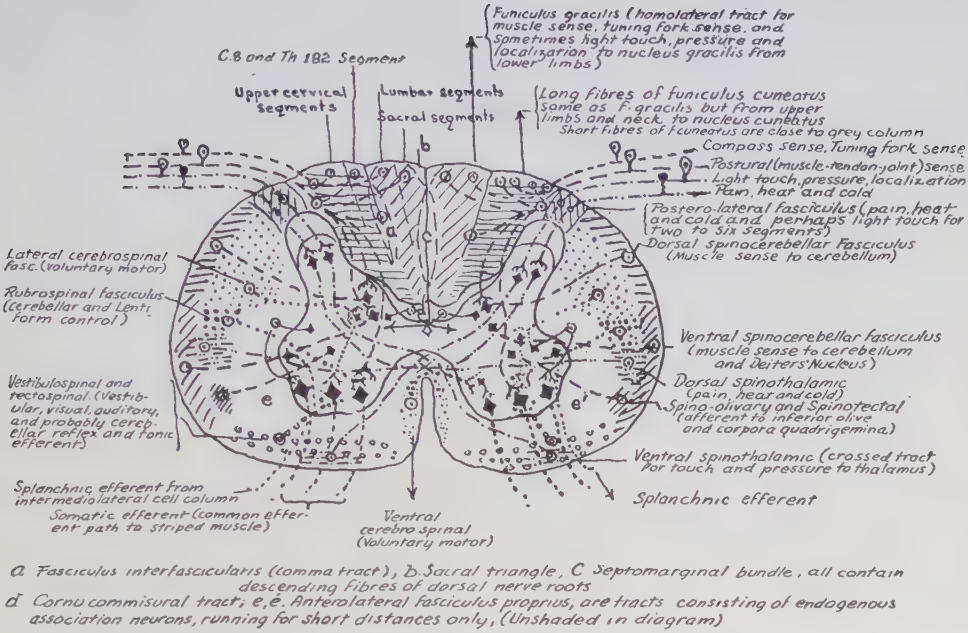

Funiculus gracilis (homolateral tract for muscle sense, tuning fork sense, and sometimes light touch, pressure and localization to nucleus gracilis from lower limbs)
C.8 and Th 1&2 Segment
Upper cervical segments
Lumbar segments
Sacral segments
Long fibres of funiculus cuneatus same as F. gracilis but from upper limbs and neck, to nucleus cuneatus
Short fibres of f. cuneatus are close to grey column
Compass sense, Tuning fork sense
Postural (muscle-tendon-joint) sense
Light touch, pressure, localization
Pain, heat and cold
Postero-lateral fasciculus (pain, heat and cold and perhaps light touch for two to six segments)
Dorsal spinocerebellar Fasciculus (Muscle sense to cerebellum)
Lateral cerebrospinal fasc. (voluntary motor)
Rubrospinal fasciculus (cerebellar and Lenti form control)
Ventral spinocerebellar fasciculus (muscle sense to cerebellum and Deiters' Nucleus)
Vestibulospinal and rectospinal. (Vestibular, visual, auditory, and probably cerebellar reflex and tonic efferent)
Dorsal Spinothalamic (pain, heat and cold)
Spino-olivary and Spinotectal (afferent to inferior olive and corpora quadrigemina)
Ventral spinothalamic (crossed tract for touch and pressure to thalamus)
Splanchnic efferent from intermediolateral cell column
Somatic efferent (common efferent path to striped muscle)
Ventral cerebro spinal (Voluntary motor)
Splanchnic efferent
a Fasciculus interfascicularis (comma tract), b. Sacral triangle, C Septomarginal bundle, all contain descending fibres of dorsal nerve roots
d Cornu commisural tract; e, ė. Anterolateral fasciculus proprius, are tracts consisting of endogenous association neurons, running for short distances only, (Unshaded in diagram)

FIG. 69.—Schema of nerves and tracts of the spinal cord. (After Cunningham, modified by W. Keiller.)

Afferent nerves and tracts indicated by broken or dot and dash lines. Efferent nerves and tracts indicated by dots and circles.

The schema represents the arrangement of the tracts in the cervical enlargement, except that the sacral triangle (b) is only found in the sacral segments and the oval field (c) is only found in the cervical and lumbo-sacral regions, both these tracts being composed of fibers descending from afferent nerves much higher in the cord. The comma tract or fasciculus interfascicularis (a) is here very conventionally indicated. Its characters and position are best seen in Fig. 80 q and 81 r and s. It also consists of descending fibers from posterior nerve roots, which occupy this position a short distance below their point of entry. The funiculus gracilis consists of long ascending fibers only. It is differentiated from the rest of the posterior column from the mid-thoracic region upward. Below this there is only one posterior column. The funiculus cuneatus is differentiated from the mid-thoracic region upward. The nerve roots enter it medially to the postero-lateral column and change their positions as they ascend or descend as the figure indicates.

The dorsolateral column is largely composed of unmyelinated fibers carrying heat, cold, and pain. This column also receives fine myelinated fibers from the posterior nerve roots which probably convey light touch, pressure, with a sense of localization. All the fibers of this column are soon relayed. The dorsal spinocerebellar tract begins at the 1st lumbar segment and increases rapidly as it ascends. The ventral spinocerebellar trace commences in the lumb-sacral region; it receives accessions all the way up. For further description see text.

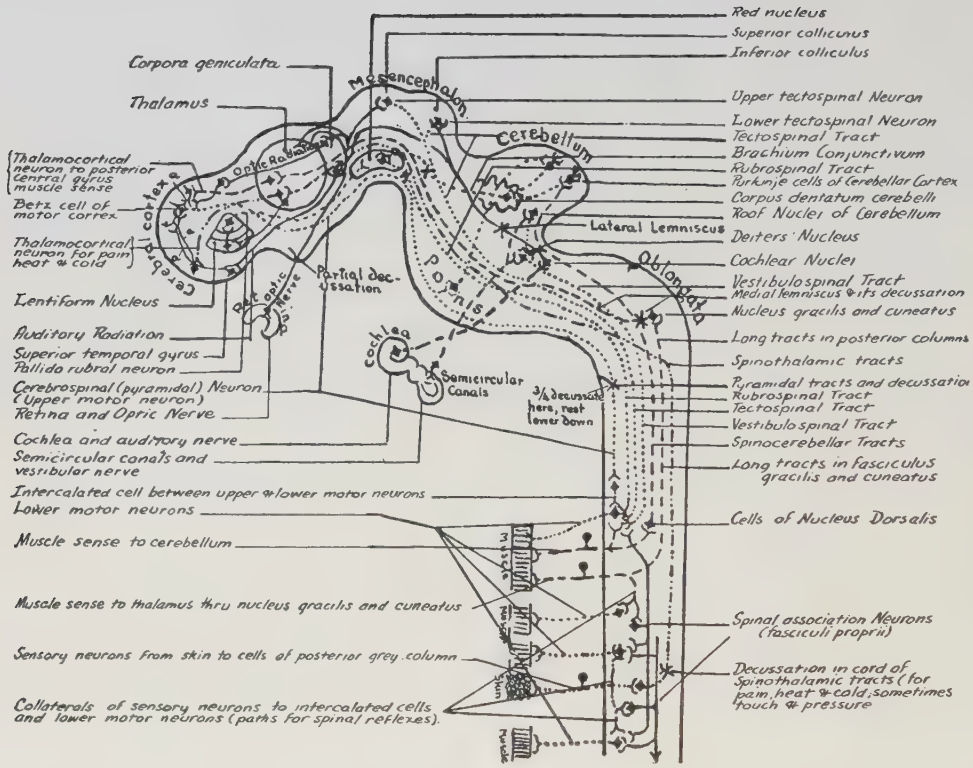


FIG. 70.—Schema of cerebro-spinal nervous system. Altered from Cunningham's Textbook. (X) indicates a decussation. (W. Keiller, 1921.) (a) Association fibers in cortex. For the sake of simplicity only one relay is shown in the thalamus.

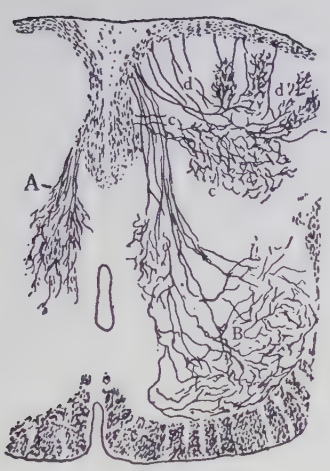


FIG. 71.

FIG. 72.

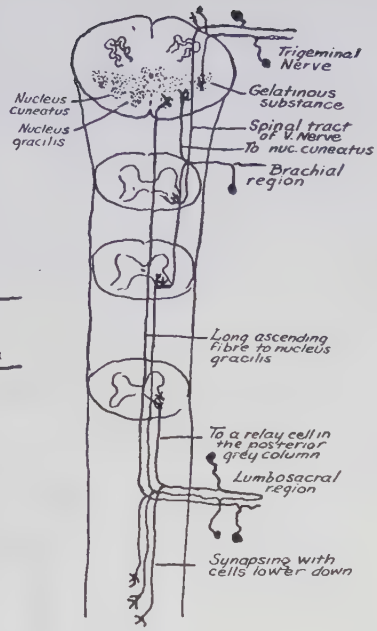


FIG. 73.

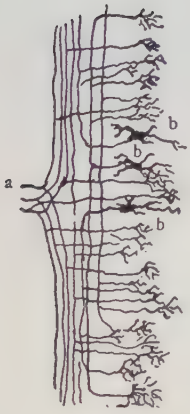


FIG. 74.

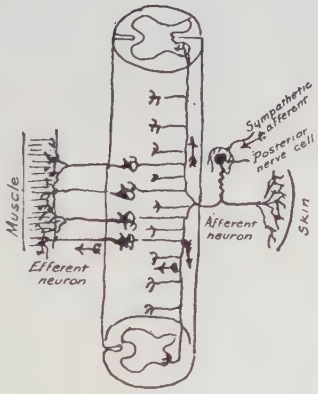


FIG. 75.

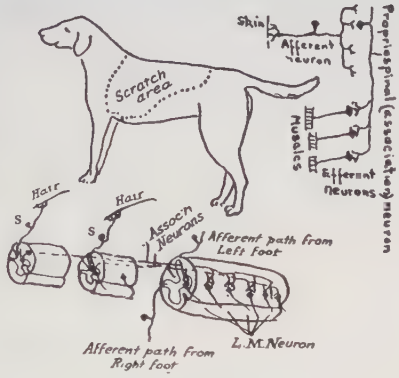


FIG. 76.

FIG. 71.—Chief collaterals of the dorsal column, newborn mouse. Gogli neurofibril stain. A, collaterals to nucleus dorsalis; B, collaterals to ventral (L.M.N.) cells; c, c, collaterals to cells of dorsal column; d, d, collaterals to cells of gelatinous substance. (Cajal.)

FIG. 72.—a, fibers of dorsal column with collaterals; b.b. to cells c.c. of gelatinous substance showing pericellular synaptic arborization. Eight day old cat. Ehrlich's method. Longitudinal section. (Cajal.)

FIG. 73.—Schema of the course of the dorsal nerve roots. (Marie and Cajal.) Each entering nerve root bifurcates into an ascending and a descending branch. Some run a short distance only and end in the gray columns of the cord. Some ascend to the nucleus gracilis or nucleus cuneatus.

FIG. 74.—Longitudinal section of cord of chick embryo. Golgi stain. a, Entering posterior nerve roots, bifurcating and sending collaterals into gray column; b, b, b, cells of endogenous neurons. (Cajal.)

FIG. 75.—Schema of a short reflex arc. (Cajal.)

FIG. 76.— Neurons involved in the scratch reflex.

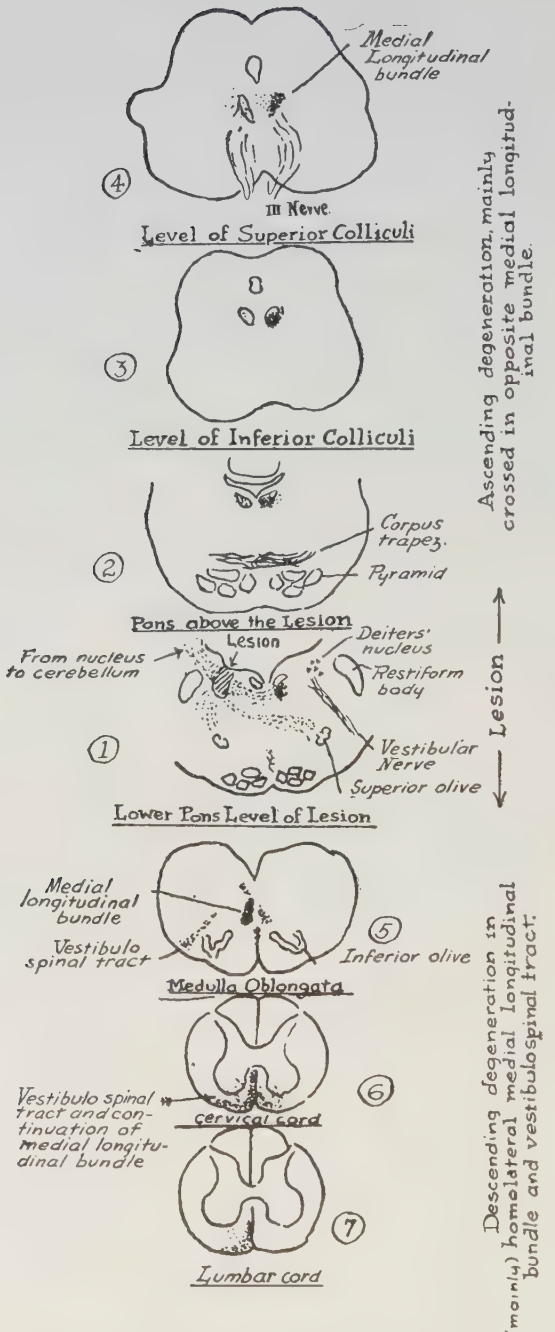


FIG. 77.—Monkey. Galvanocautery destruction of Deiters' nucleus. Marchi stain. (From E. H. Fraser, Jour. Phys., 1901-02.)

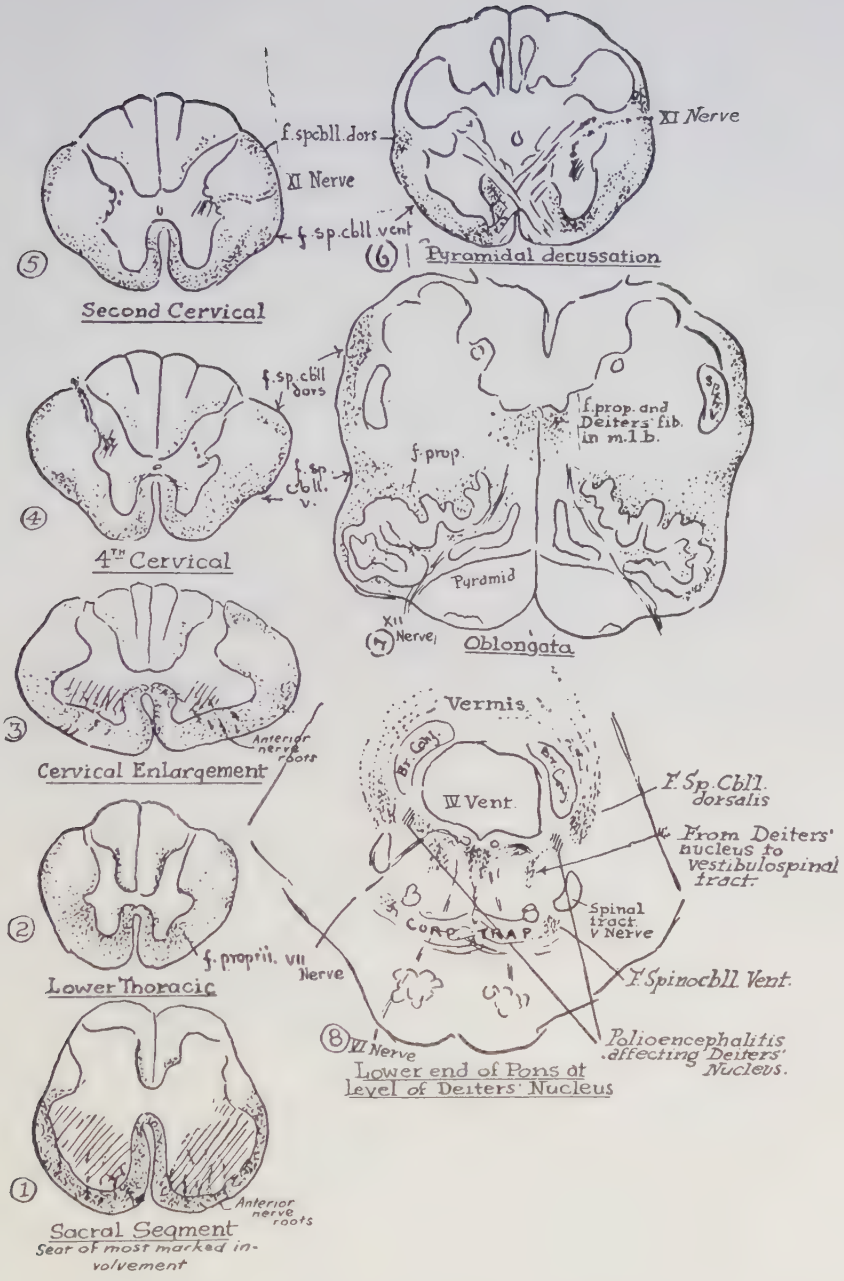


FIG. 78.—Endogenous fibers of human spinal cord. From a case of poliomyelitis, chiefly in the lumbar segments, but also in the cervical enlargement, and affecting smaller areas higher up, including nuclei of the XI nerve and both the nuclei of Deiters'. Oblique shading shows disease. Dots show degenerated fibers. (Batten and Holmes; Brain, 1912.)

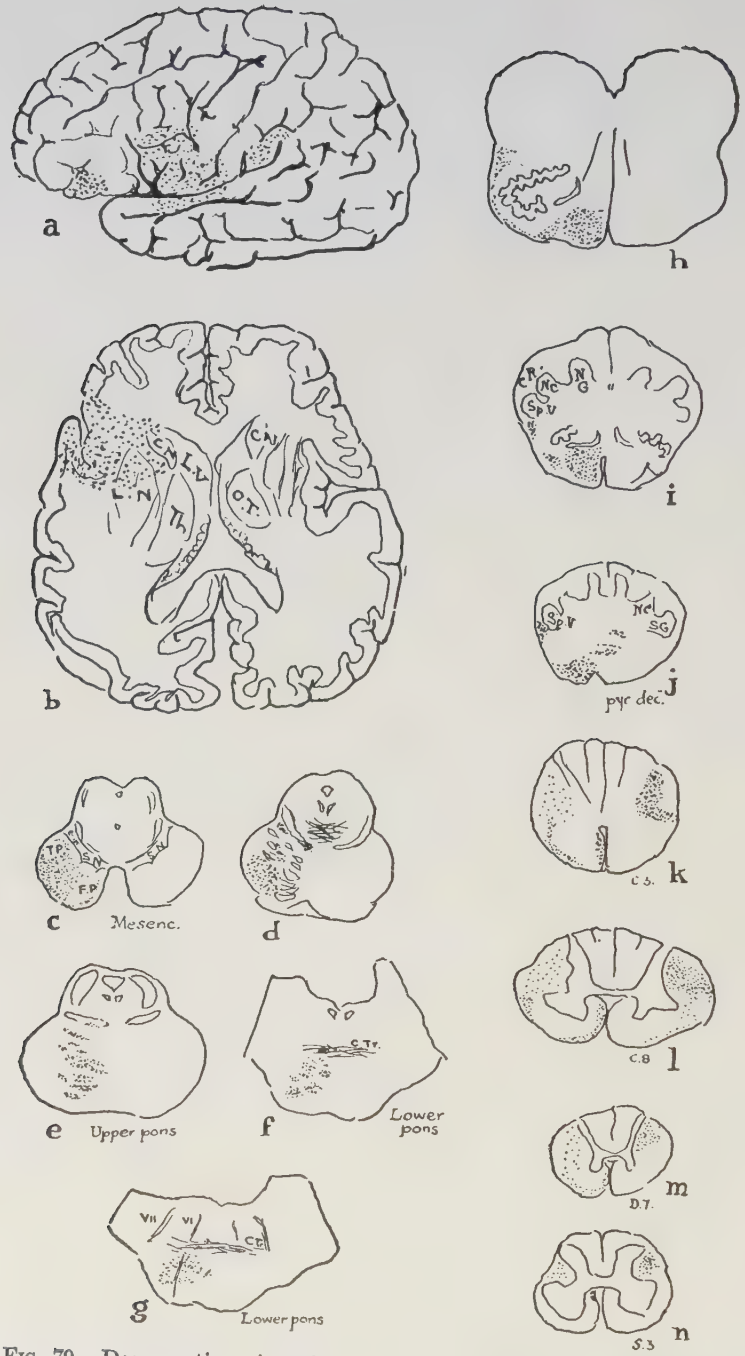


FIG. 79.—Degenerations in softening of cerebral cortex by occlusion of branches of the middle cerebral artery. Marchi stain. (S. Barnes, Brain, XXIV.)

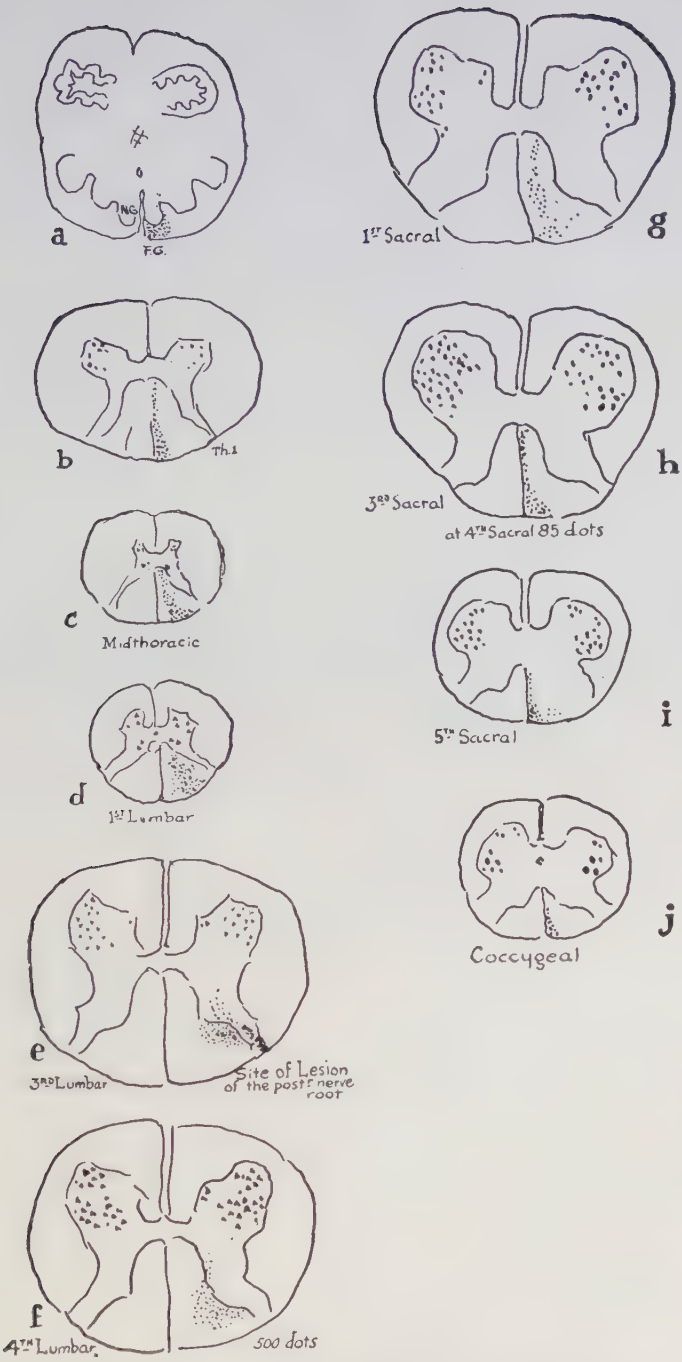


FIG. 80.—Compression of 3rd lumbar nerve root by carcinoma of spinal column. Ascending and descending degenerations shown by Marchi stain. (Collier and Buzzard; Brain, XXVI, 1903.)

Note how both ascending and descending fibers approach the median septum as they ascend and descend respectively.

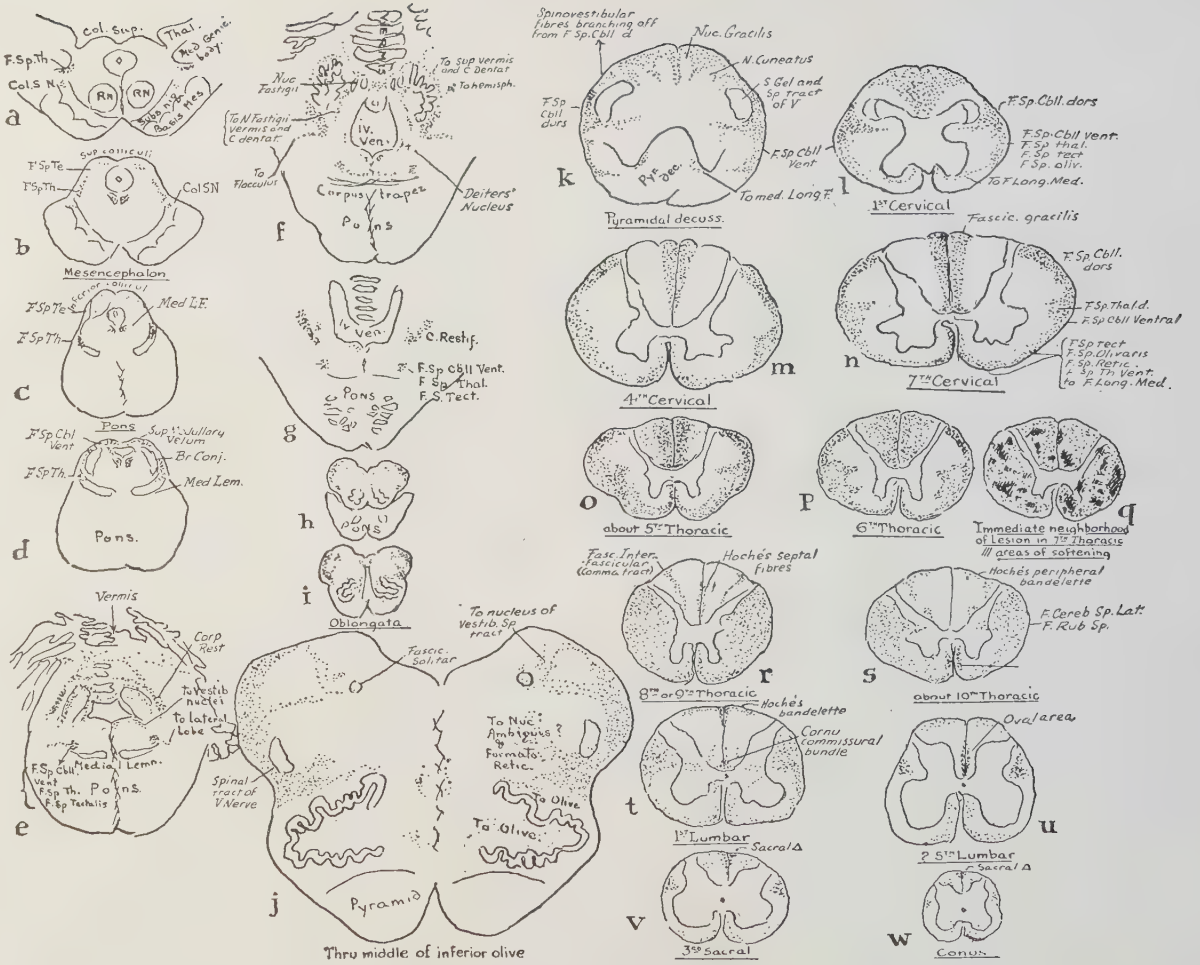


FIG. 81.—Degenerations in a complete transverse lesion of the spinal cord in man. Marchi stain. (Collier and Buzzard; Brain, 1903.)

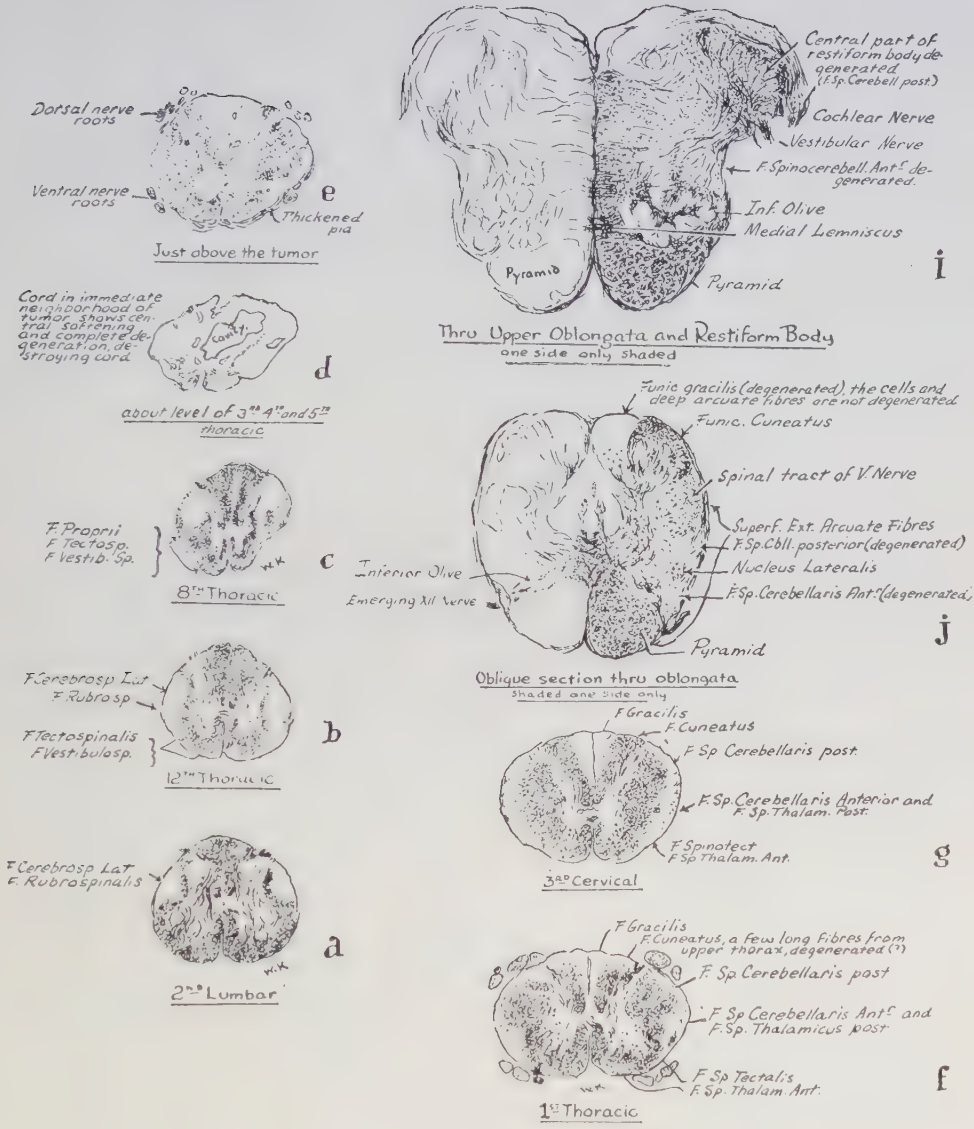


FIG. 82.—Degenerations of cord in case of tumor of dura, causing complete interruption of tracts at level of 3rd, 4th, and 5th thoracic segments. Shows ascending and descending degeneration as brought out by Pal-Weigert hematoxylin, which stains myelin-sheath purple. Degenerated areas unstained. For tumor mass see Fig. 185. (Lab. of Anat., U. of Tex., W. Keiller.)

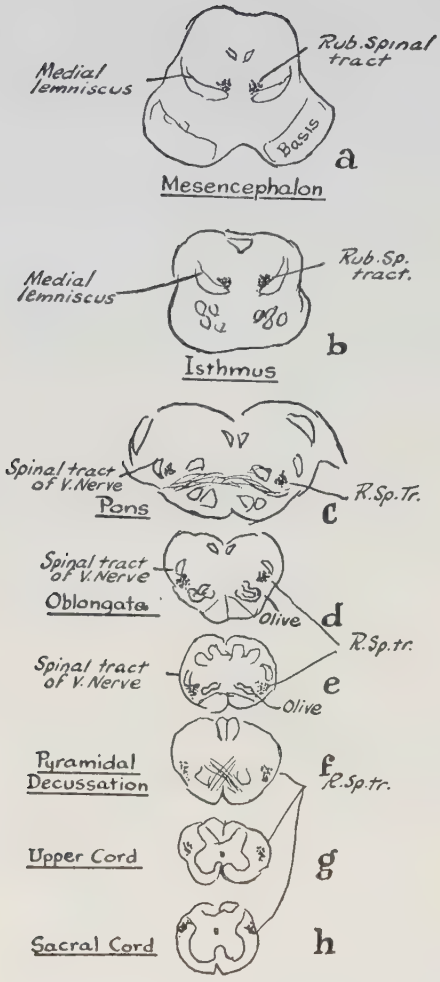


Fig. 83.—Degeneration of the rubrospinal tract after bilateral destruction of the red nuclei in an ape. Rubrospinal tract, Marchi stain. (From Collier and Buzzard, Edinger.)

Fig. 84.—Atrophy of left inferior olive in child born without the right cerebellar hemisphere. (Edinger.)

Under surface of cord in lumbar enlargement showing position and extent of section that would cut the posterior spinal thalamic tract.

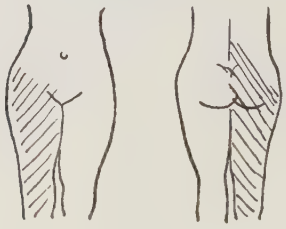


Fig. 85.—Patient with inoperable pelvic cancer causing extreme pain all over right leg. Section of posterior spino-thalamic tract on the left side at the middle of the lumbar enlargement. Complete relief of pain. No interference with nutrition or function of limb.

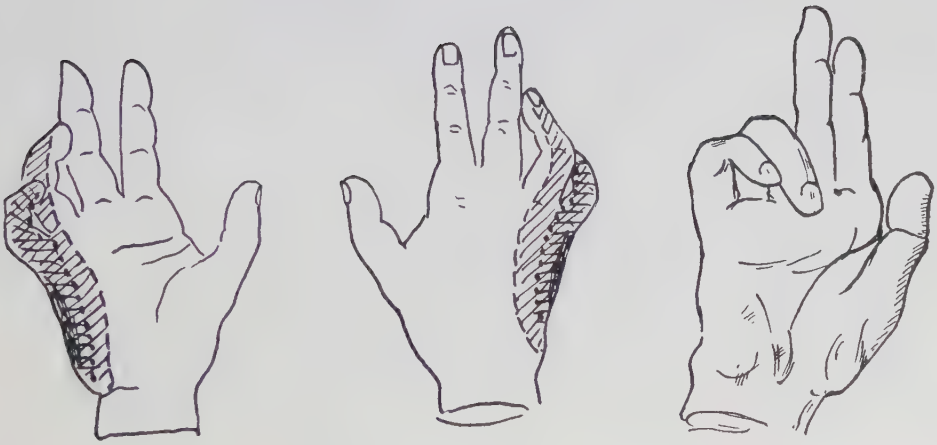


Fig. 86. Fig. 86b.

Fig. 86.—Abnormal position due to paralysis of interossei and lumbricals and loss of sensation after section of the ulnar nerve just above the wrist. Oblique lines: loss of epicritic sense. Horizontal lines: loss of protopathic sense. Deep shading: loss of deep sensibility. (Bowlby, Starr.)

Fig. 86b.—Shows wasting and deformity in later stage of ulnar paralysis.

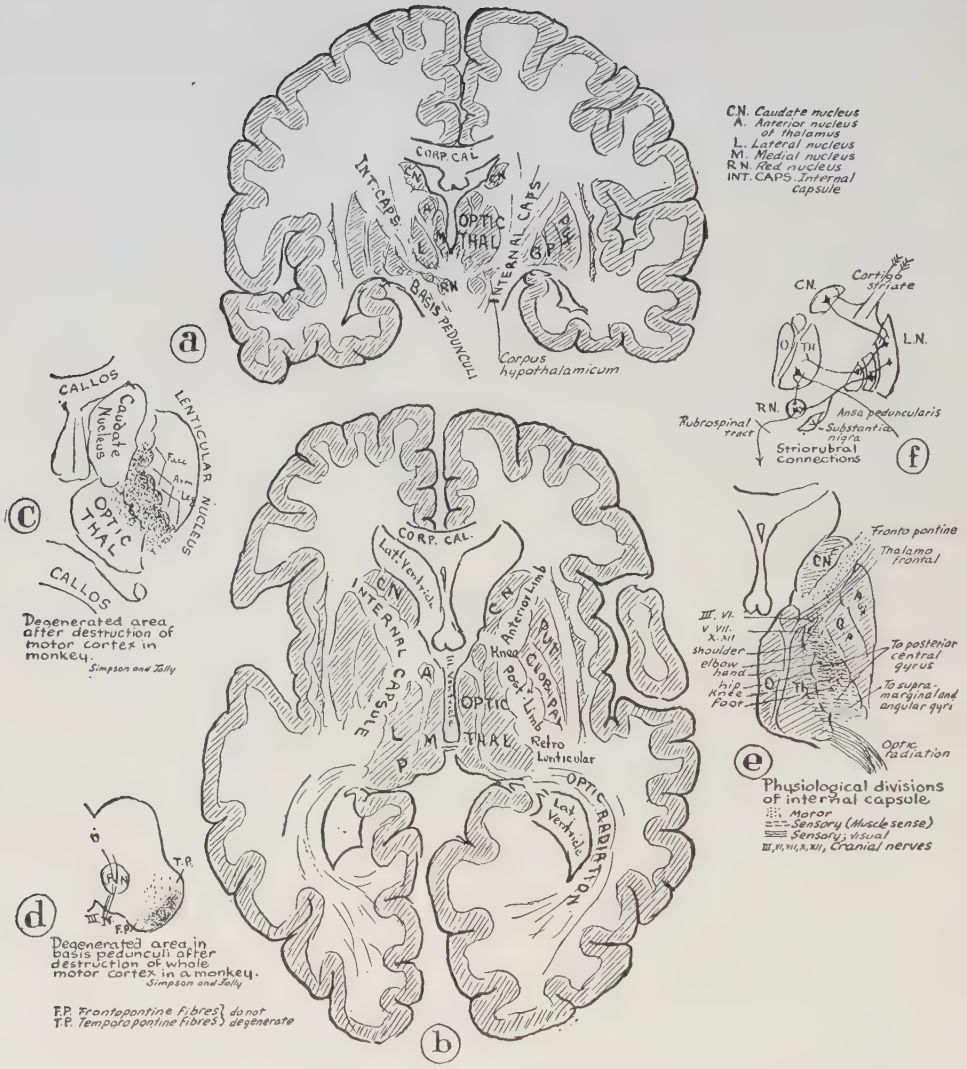


FIG. 87.—a, b, Vertical and horizontal sections of brain to show basal ganglia, thalamus and internal capsule; c, d, degenerations as shown by Marchi stain after experimental lesions in a monkey; e, schematic divisions of internal capsule; f, connections of basal ganglia and thalamus.

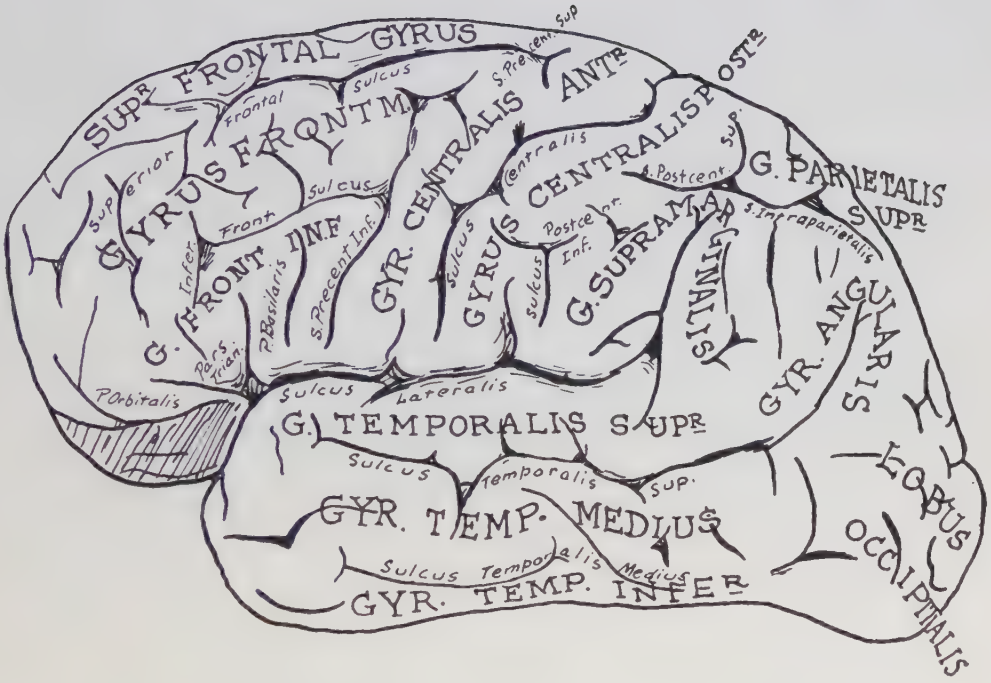


FIG. 88.—Convolutions of the lateral surface of a human brain, simple type. (After Symington in Quain's Anatomy.)

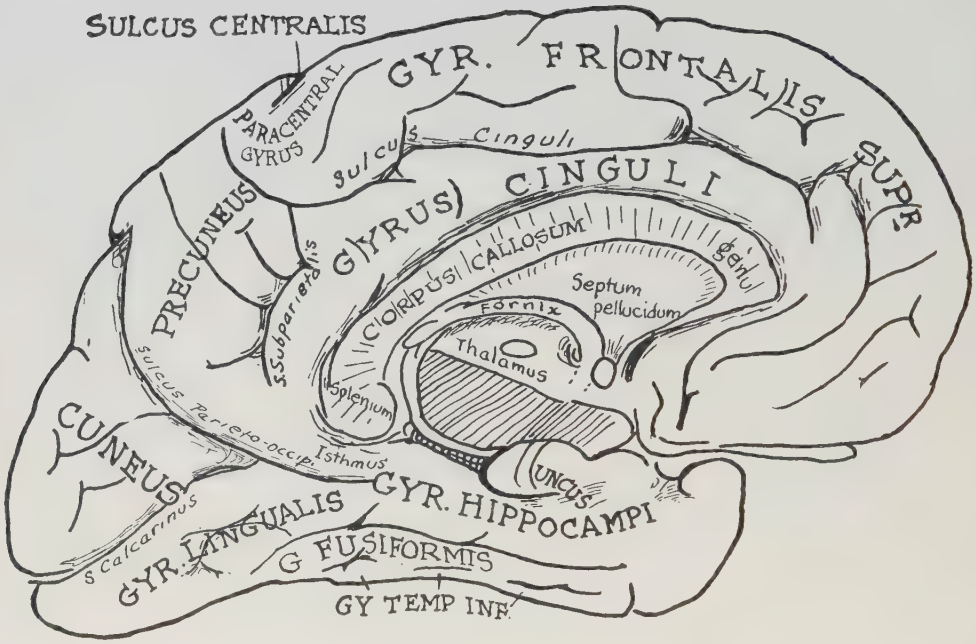


Fig. 89.—Convolutions of the medial surface of a human brain, simple type, (After Symington in Quain's Anatomy.)

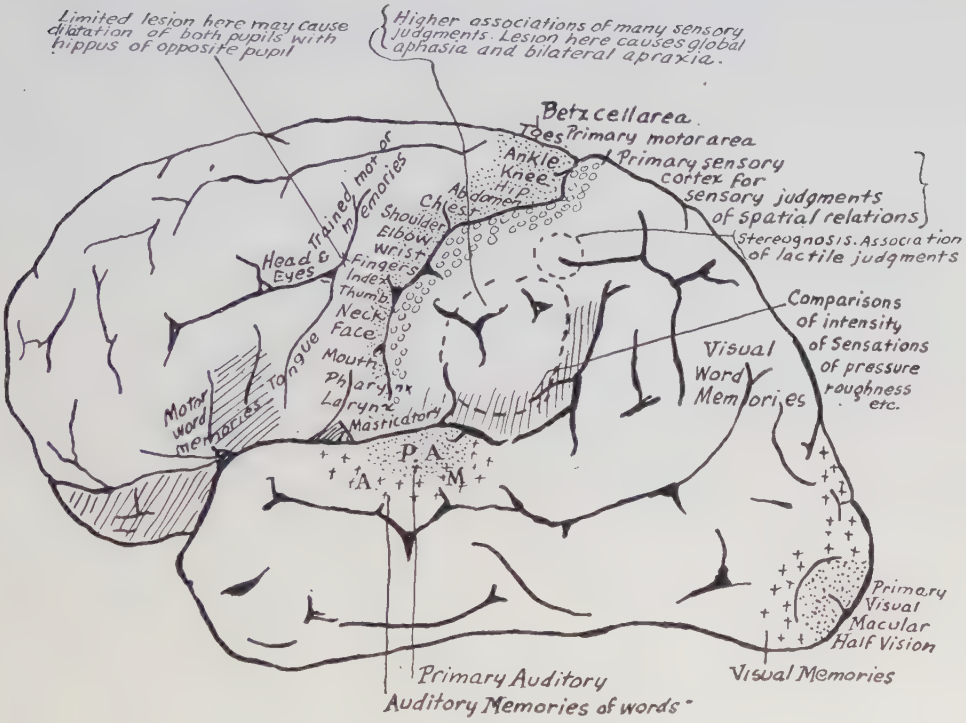


FIG. 90.—Motor, sensory, and other localizing areas of cerebral cortex compiled from various sources. (W. Keiller.)

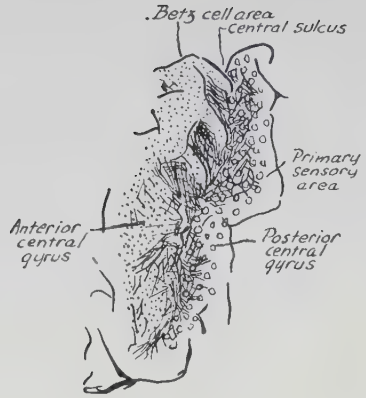


Fig. 91.

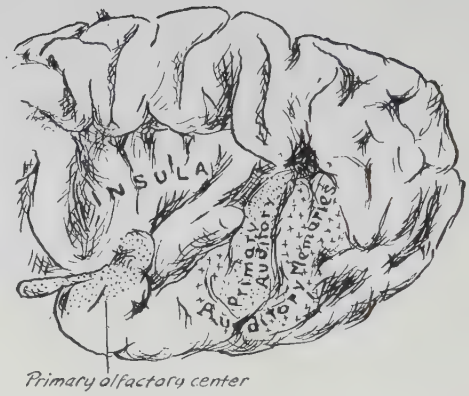


Fig. 92.

Figs. 91 and 92.—Primary and secondary motor and sensory areas of cerebral cortex. Fig. 91—in the walls of the central sulcus; Fig. 92—in the superior temporal convolution as shown when the opercula are separated. (After Cunningham.)

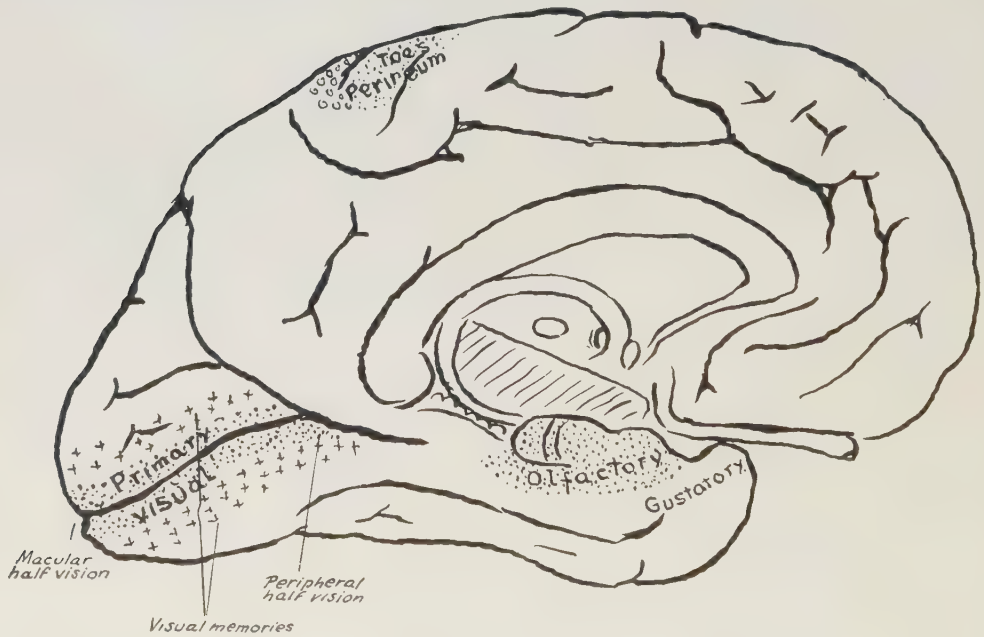


Fig. 93.—Motor and sensory areas on the medial surface of the human brain.

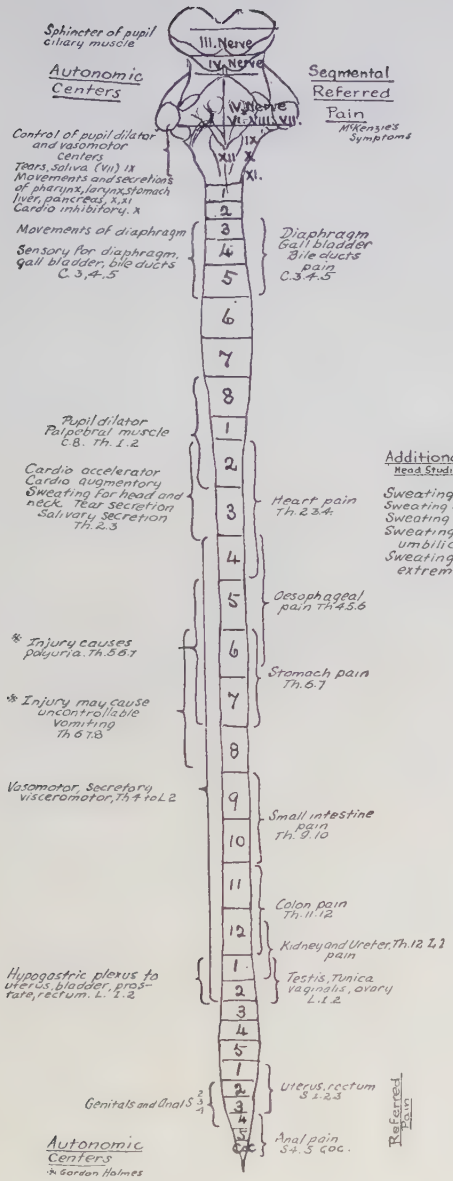

Sphincter of pupil
Ciliary muscle
III. Nerve
IV. Nerve
Autonomic Centers
Segmental Referred Pain
Mackenzie's Symptoms
V. Nerve
VI. XIII. VIII.
Control of pupil dilator and vasomotor centers
Tears, saliva (VII) IX
Movements and secretions of pharynx, larynx, stomach liver, pancreas, X, XI
Cardio inhibitory. X
Movements of diaphragm
Sensory for diaphragm, gall bladder, bile ducts C.3,4.5
Diaphragm Gall bladder Bile ducts pain C.3.4.5
Pupil dilator Palpebral muscle C.8. Th.1.2
Cardio accelerator Cardio augmentory Sweating for head and neck. Tear secretion Salivary secretion Th.2.3
Heart pain Th.2.3.4
Oesophageal pain Th.4.5.6
* Injury causes polyuria. Th.5.6.7
* Injury may cause uncontrollable vomiting Th.6.7.8
Stomach pain Th.6.7
Vasomotor, Secretory visceromotor, Th.7 to L.2
Small intestine pain Th.9.10
Colon pain Th.11.12
Kidney and Ureter Th.12, L.1 pain
Hypogastric plexus to uterus, bladder, prostate, rectum. L. 1.2
Testis, Tunica vaginalis, ovary L.1.2
Genitals and Anal S.2.3.4
Uterus, rectum S.1.2.3
Anal pain S.4.5 Coc.
Referred Pain
Autonomic Centers
* Gordon Holmes

FIG. 94.

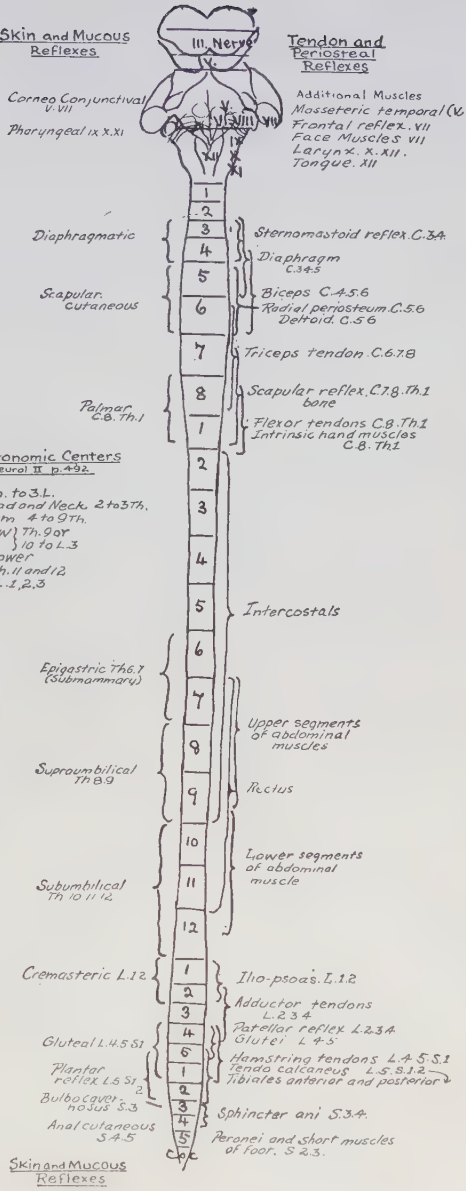


FIG. 95.

FIGS. 94 and 95.—Reflex centers of human cord and brain stem. In Fig. 95, the segmental supply of some of the chief localizing muscles is also shown.

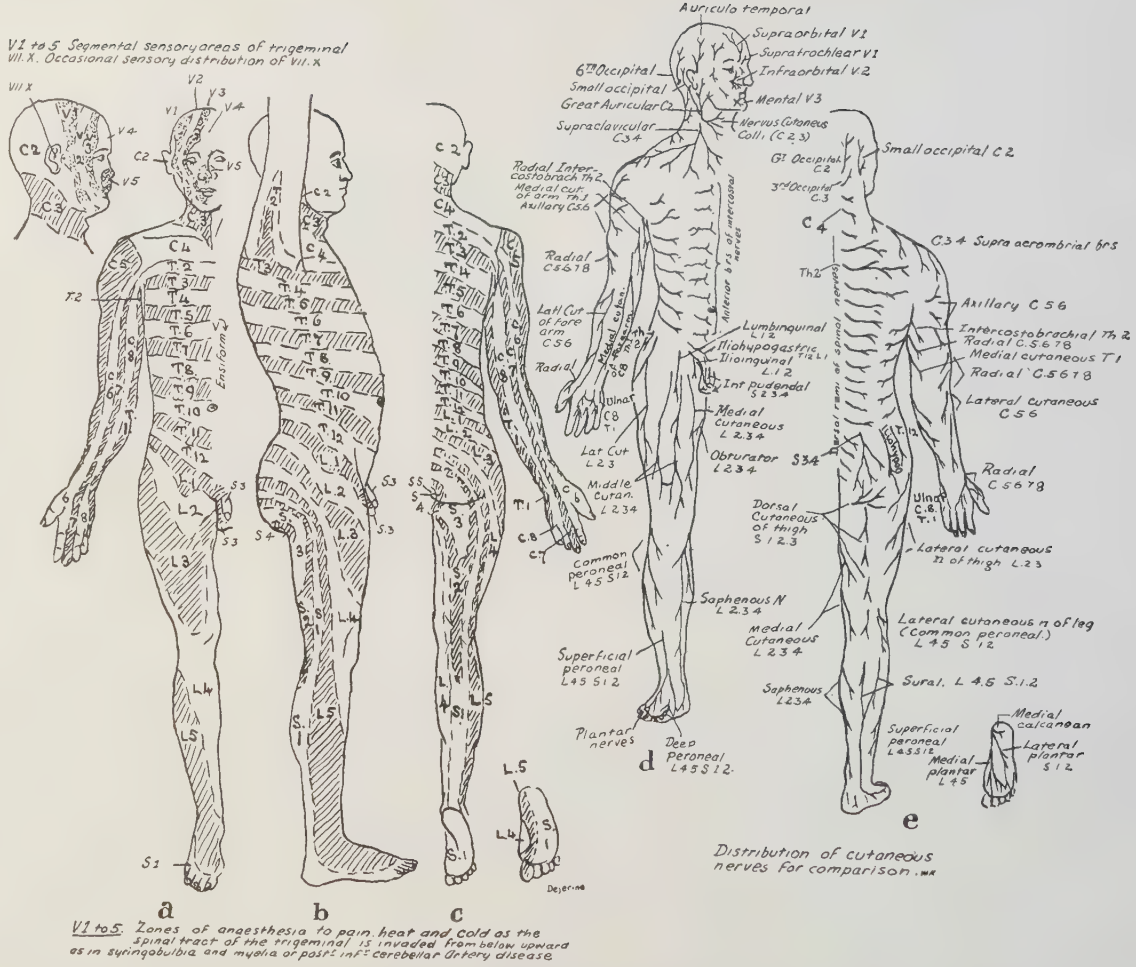

V1 to 5 Segmental sensory areas of trigeminal
VII. X. Occasional sensory distribution of VII. X
a
b
c
V1 to 5. Zones of anaesthesia to pain, heat and cold as the spinal tract of the trigeminal is invaded from below upward as in syringobulbia and myelia of post. inf. cerebellar artery disease.
Auriculo temporal
Supraorbital V1
Supratrochlear V1
Infraorbital V2
Mental V3
Nervus Cutaneus Colli (C2 3)
6th Occipital
Small occipital
Great Auricular C2
Supraclavicular C34
Radial Intercostobrach Th2
Medial cut. of arm Th1
Axillary C5 6
Radial C5 6 7 8
Lat Cut of fore arm C5 6
Ulnar C8 T1
Lat Cut L 2 3
Middle cutan. L 2 3 4
Common peroneal L 4 5 S 1 2
Superficial peroneal L 4 5 S 1 2
Plantar nerves
Deep Peroneal L 4 5 S 1 2
Anterior br. of intercostal nerve
Lumbinguinal L 1 2
Iliohypogastric T 12 L 1
Ilioinguinal L 1 2
Int pudendal S 2 3 4
Medial cutaneous L 2 3 4
Obturator L 2 3 4
Saphenous N L 2 3 4
Medial Cutaneous L 2 3 4
Saphenous L 2 3 4
d
Small occipital C 2
Gt Occipital C 2
3rd Occipital C 3
C 3 4 Supra acrombrial brs
Axillary C 5 6
Intercostobrachial Th 2
Radial C 5 6 7 8
Medial cutaneous T 1
Radial C 5 6 7 8
Lateral cutaneous C 5 6
Radial C 5 6 7 8
Lateral cutaneous n of thigh L 2 3
Lateral cutaneous n of leg (Common peroneal) L 4 5 S 1 2
Sural L 4 5 S 1 2
Medial calcanean
Lateral plantar S 1 2
Medial plantar L 4 5
Superficial peroneal L 4 5 S 1 2
e
Distribution of cutaneous nerves for comparison.

FIG. 96.—a, b, c, Skin areas (dermatomes) corresponding to the dorsal nerve roots. (Holmes and Walshe in Nelson's Loose Leaf Medicine.) d, e, distribution of cutaneous nerves for comparison. (W. Keiller.)

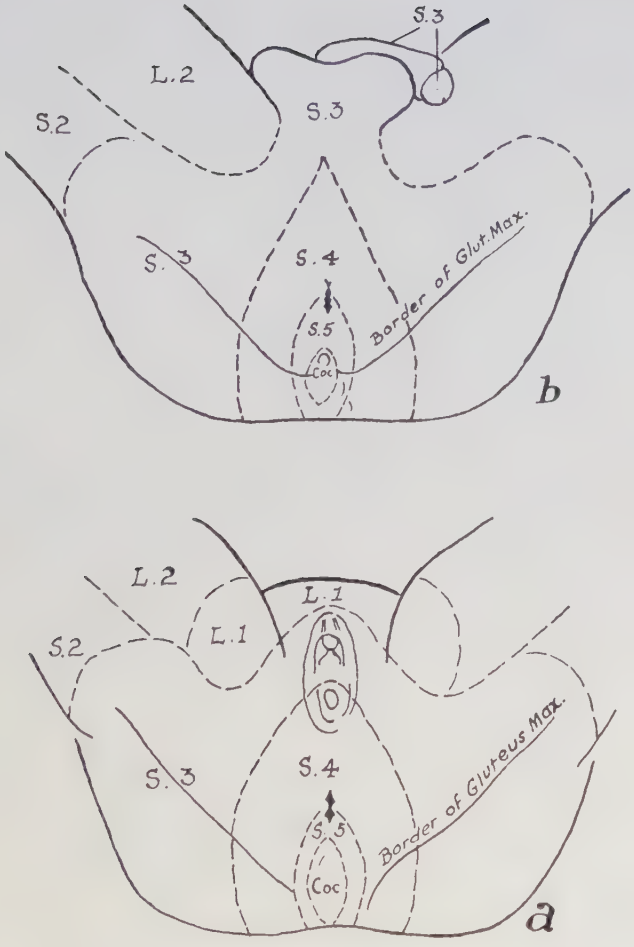


FIG. 97.—Sensory areas in male and female perineum with the segments of cord representing them. (After Dejerine.)

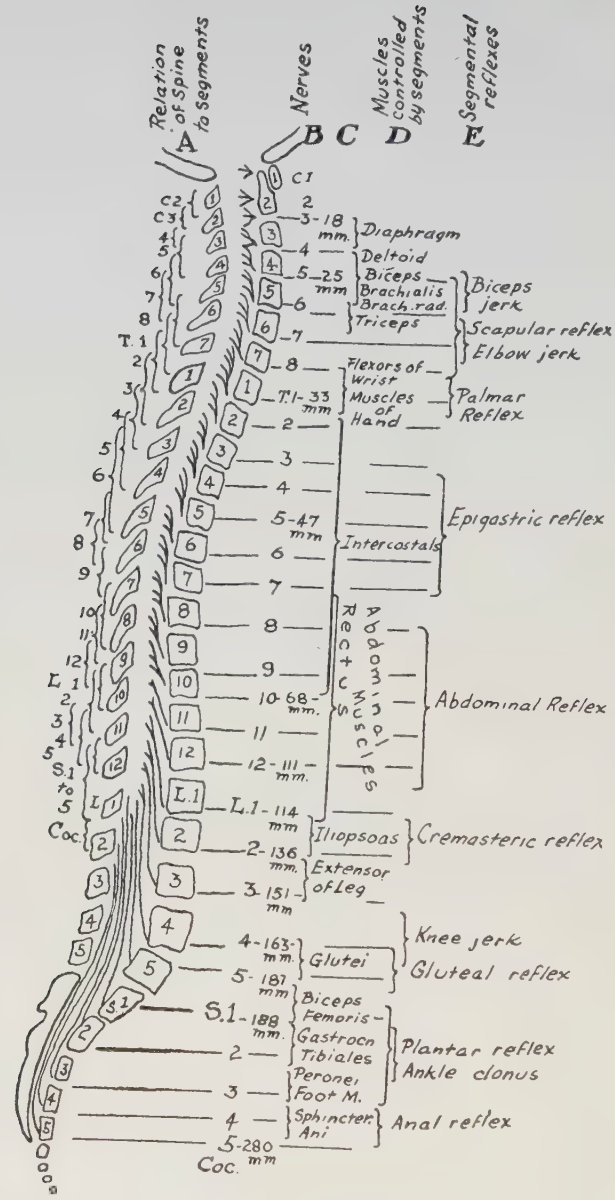


FIG. 98.—Topography of the segments of the spinal cord. Column A, extreme limits of relations of cord segments to spine (Reid); Column B, shows each nerve cut off at its intervertebral foramen, thus illustrating the obliquity of its course in the spinal canal; Column C, vertical distance in millimeters which, in Testut's case of a subject of 18 years, separated the superficial origin of each of the nerves marked from its intervertebral foramen; Column D, muscles supplied by each segment most useful for clinical purposes; Column E, segmental skin and muscle reflexes. (W. Keiller, in Anatomy by American Authors, modified.)

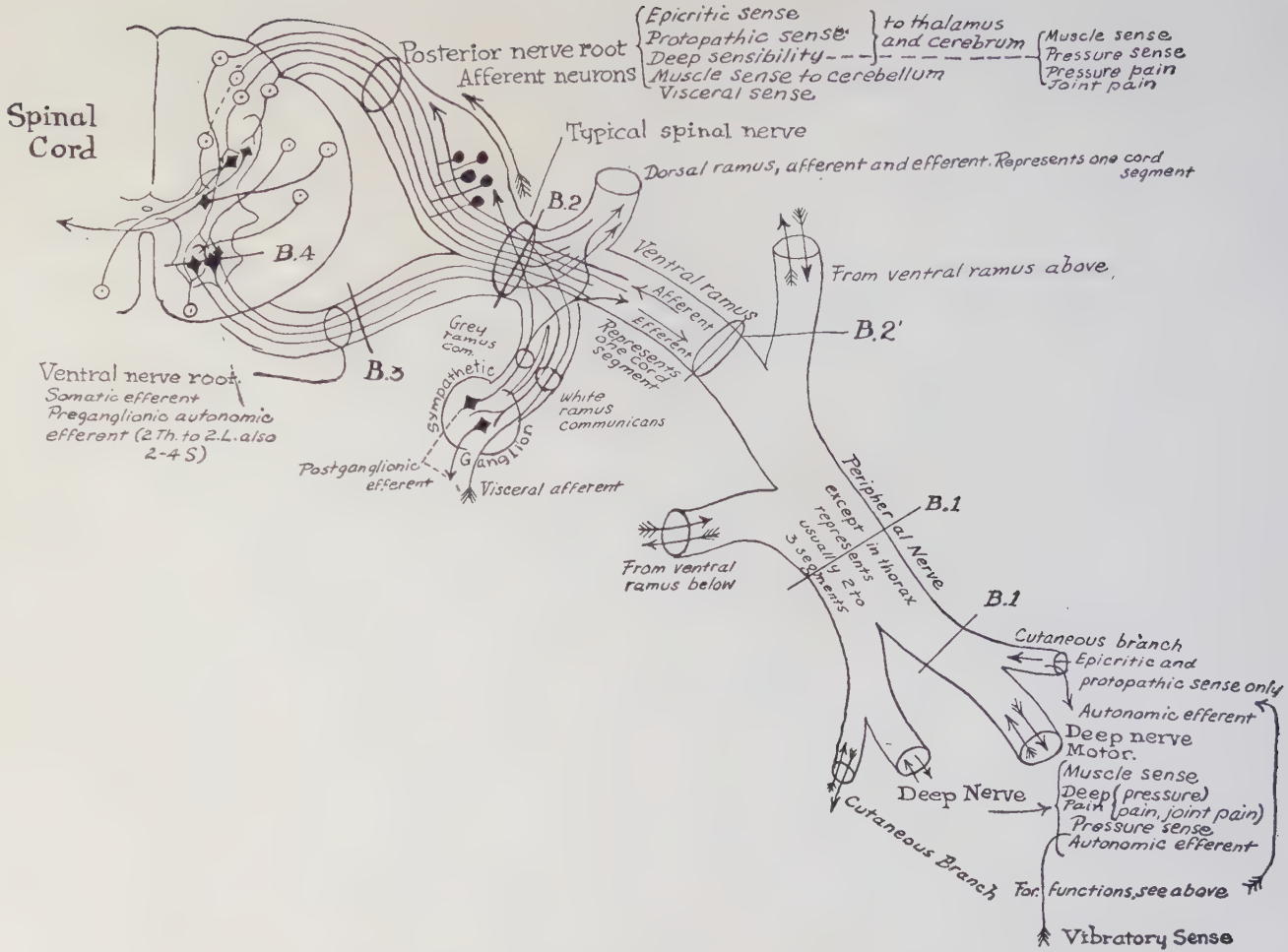


FIG. 99.—Diagram of a typical spinal nerve. B.1 to B.4 typical lesions. See page 294.

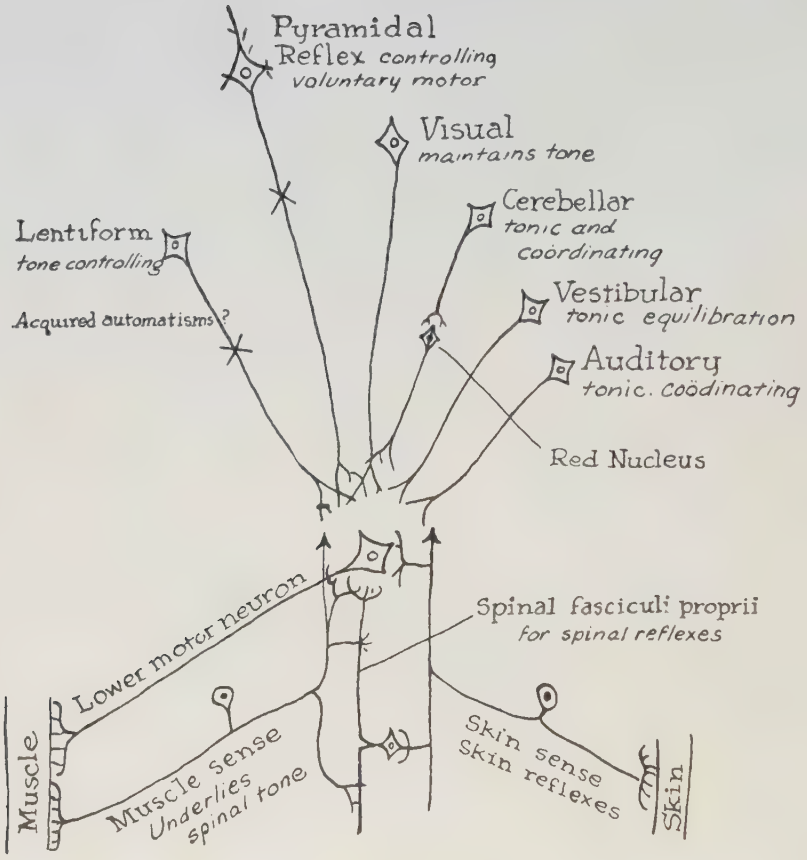


FIG. 100.—Diagram of nervous influences underlying muscle tone.

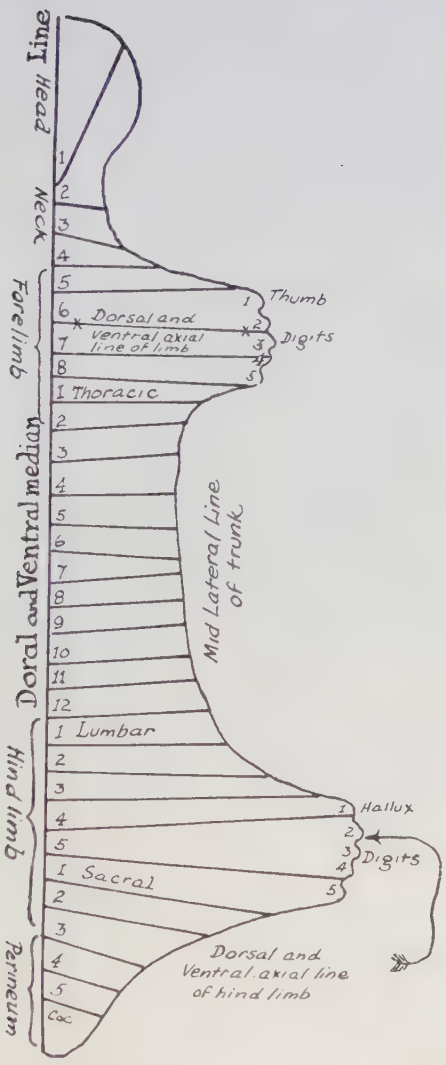


FIG. 101.—Segmental arrangement of cutaneous areas.

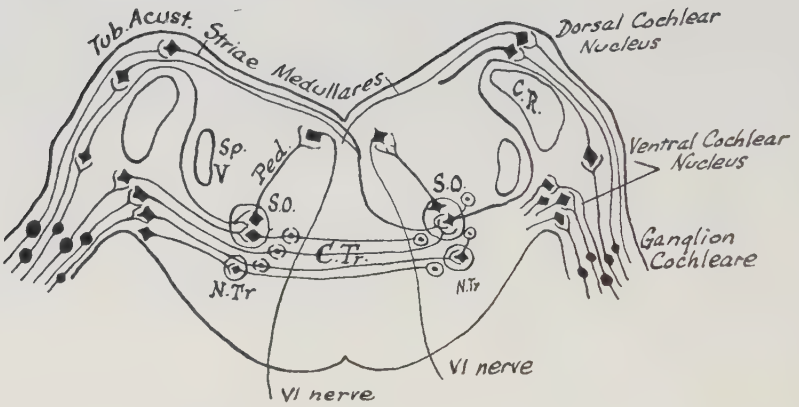


FIG. 102.—Diagram of the cochlear nerve and its connections in the pons. C. R., corpus restiforme; Sp. V., spinal tract of V nerve; C. Tr., corpus trapezoideum; S. O., superior olive; N. Tr., nucleus of corpus trapezoideum; Ped., peduncle of superior olive. Circles with central dots show where the fibers of the corpus trapezoideum become vertical (*i.e.*, longitudinal), forming the lateral lemniscus.

Fig 103a. 3rd Lumbar

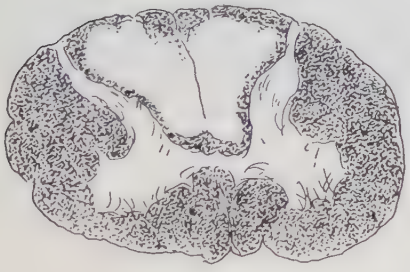


Fig 103b. 5th Cervical

FIG. 103.—Sections of cord stained by Pal-Weigert method, showing primary sclerosis of posterior columns. Symptoms: ataxia, loss of sense of position, impaired sense of weight, position, impaired sense of weight, loss of tactile discrimination. (Thompson, Brain, XXXIV.)

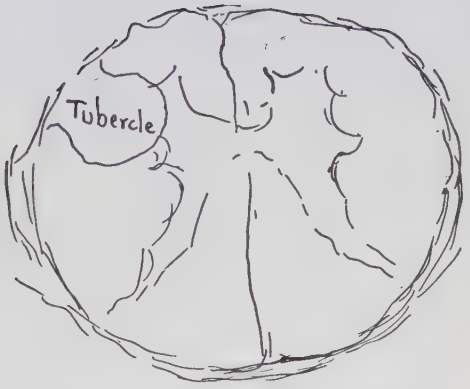


Fig. 104a. Tubercle at extreme lower end of thoracic cord.

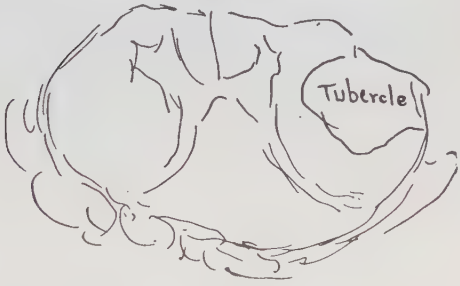


Fig. 104b. Tubercle in left lateral column $\frac{1}{2}$ inch higher than the tubercle in fig. 104a.

FIG. 104.—Spiller's case of two separate tubercles in same cord interrupting both posterior spinothalamic tracts. (Univ. Penna. Med. Bul., 1905.)

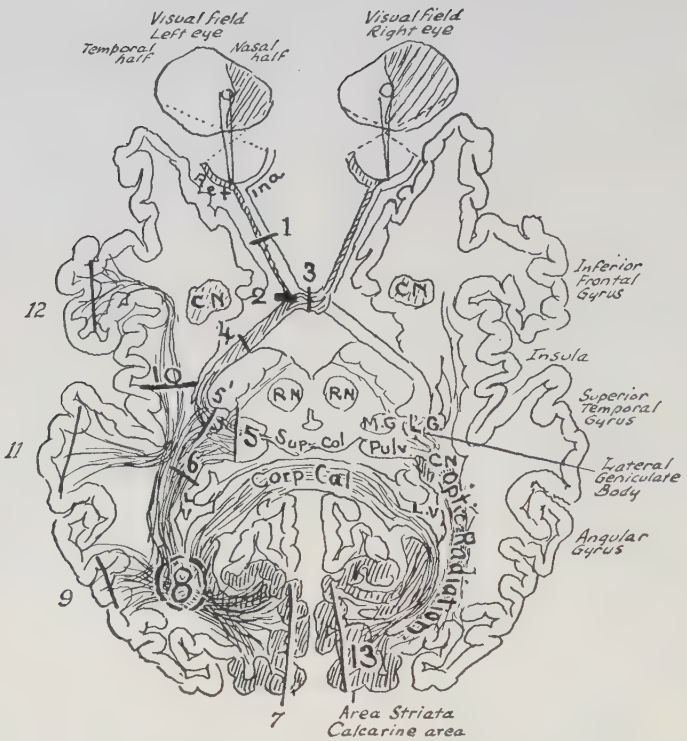


FIG. 105.—Projection paths for the optic nerves with typical lesions. (Dejerine, modified.) C. N., caudate nucleus; R. N., red nucleus; M. G., medial geniculate body; L. G., lateral geniculate body; Sup. Col., superior colliculi; Corp. Cal., corpus callosum; Pulv., pulvinar. Typical lesions: 1, blindness of left eye; 2, left nasal hemianopsia; 3, bitemporal hemianopsia; 4, right homonomous hemianopsia; 5^1, 5, 6, same as 4; 7, right homonomous hemianopsia (cortical type); 8, ditto with word blindness (in right-handed patient); 9, aphasia (angular type); 10, aphasia (complex forms) including alexia; 11, sensory aphasia (temporal type); 12, motor aphasia (frontal type).

7 and 13, cortical blindness of both fields.

6, 7, 8, hemianopsia with normal pupillary reaction (pupils contract to light).

2, 3, 4, 5^1, 5, hemianopsia with Wernicke's pupillary reaction. Light stimulation of blind field fails to give pupillary reaction.

Note: A pure lesion of the pulvinar (5) does not cause hemianopsia, but a pure lesion of the lateral geniculate body (5^1) does.

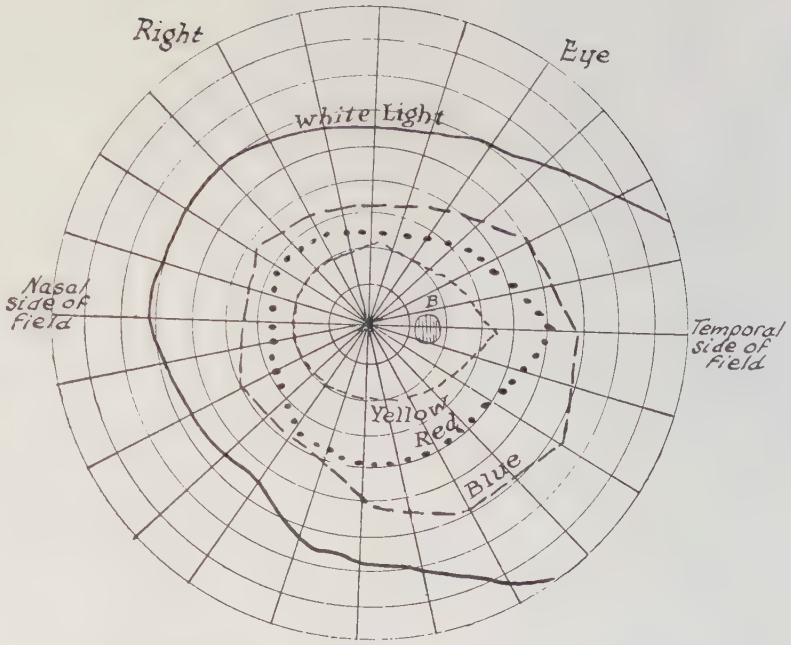


Fig. 106.—Normal visual field for right eye as outlined on perimetric chart. For white light, blue, red and yellow. B, blind spot (optic disc). (Barker, Monographic Medicine, Vol. I, page 165.)

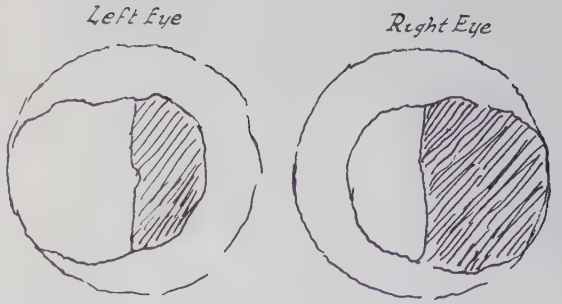


Fig. 107.—Right homonomous hemianopsia from softening of left occipital lobe. (Stewart.)

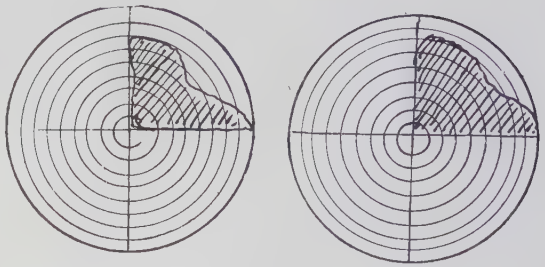


Fig. 108.—Right quadrantal hemianopsia for colors, without limitation of field for white light. Gunshot wound of left occipital lobe below calcarine fissue. (Brain, XXXIX.)

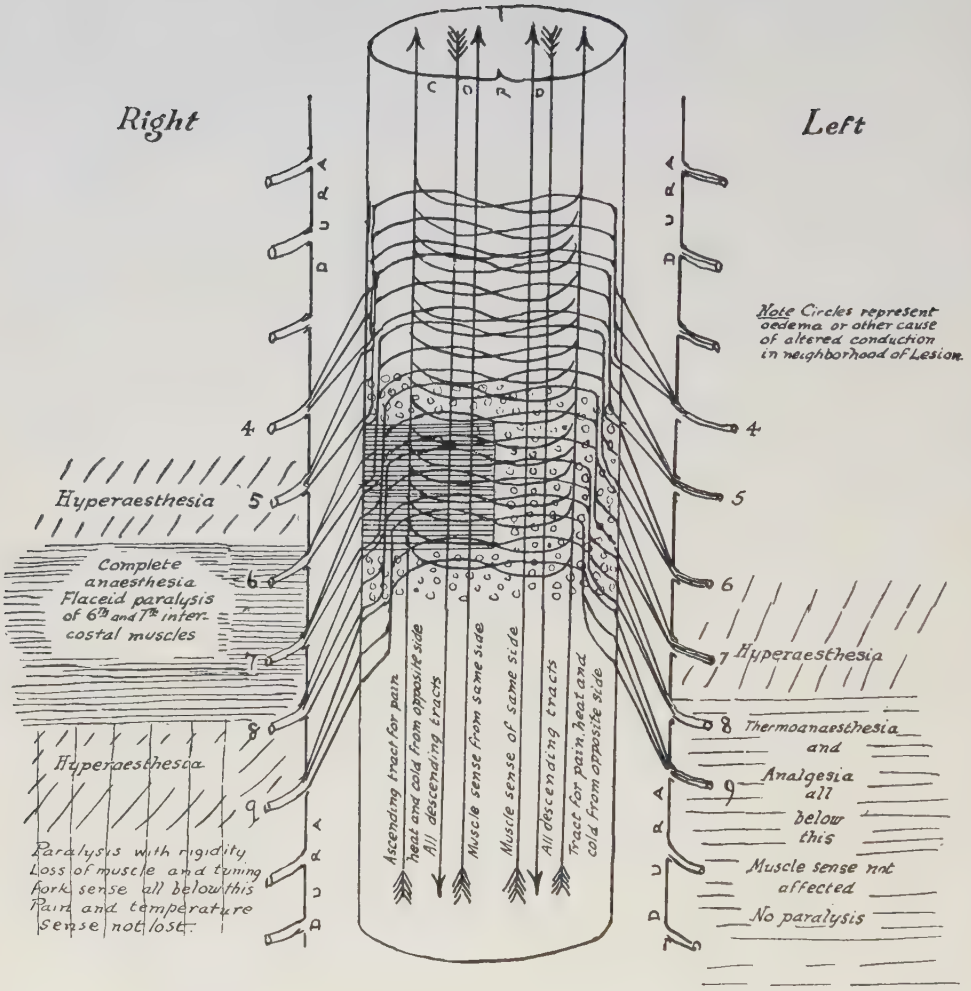

Right
Left
Note Circles represent oedema or other cause of altered conduction in neighborhood of Lesion.
Hyperaesthesia
Complete anaesthesia Flaccid paralysis of 6th and 7th intercostal muscles
Hyperaesthesia
Paralysis with rigidity Loss of muscle and tuning fork sense all below this Pain and temperature sense not lost.
Ascending tract for pain heat and cold from opposite side
All descending tracts
Muscle sense from same side
Muscle sense of same side
All descending tracts
Tract for pain heat and cold from opposite side
Hyperaesthesia
Thermoanaesthesia and Analgesia all below this
Muscle sense not affected
No paralysis

FIG. 109.—Illustrating Brown Sequard paralysis. Half section of cord. Shaded area indicates lesion. As applied to case illustrated in Fig. 110, the diagram represents the cord as seen from the front.

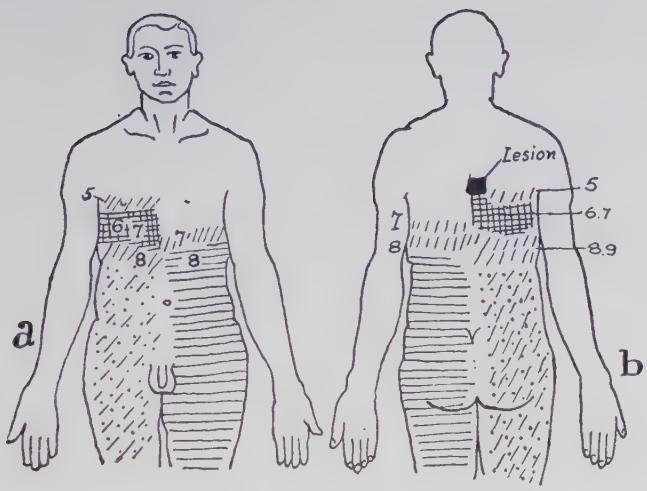


FIG. 110.—Case of Brown Sequard paralysis from half lesion of cord in 6th and 7th thoracic segments on the right side. Oblique lines, hyperæsthesia; horizontal lines, analgesia, thermoanæsthesia; dots, spastic paralysis; criss-cross shading, complete anæsthesia, flaccid paralysis, short oblique lines, slight hyperæsthesia.

Compare with Fig. 41, where the lesion of the cord is seen as viewed from the front. For segmental areas compare Fig. 96.

FIG. 111.—Site of glioma (syringomyelia) of 7th and 8th cervical and 1st thoracic segments of cord causing symptoms illustrated in Fig. 112.

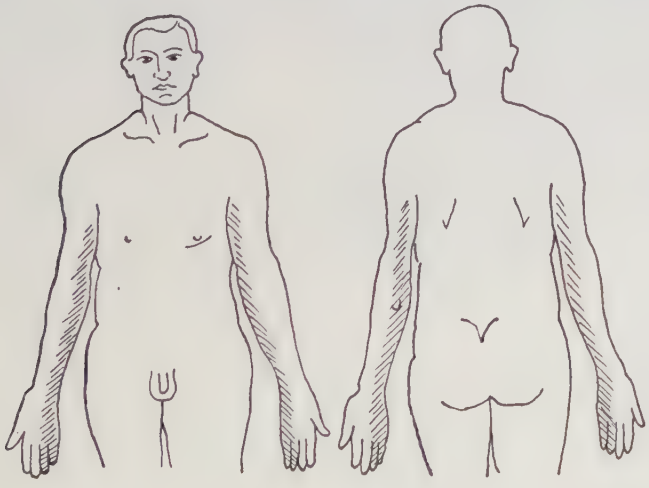


FIG. 112.—Distribution of thermoanæsthesia and analgesia; case of syringomelia involving the anterior commissure in the 7th and 8th cervical and 1st thoracic segments of the spinal cord. See Fig. 111.

FIG. 113.—Case of syringomyelia with multiple foci; shows areas of thermanæsthesia and analgesia corresponding to segments C, 2, 3, 4, 5 and L, 3. Compare with Fig. 96.

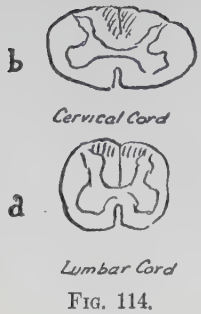


Fig. 114.

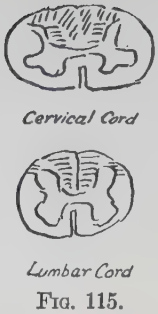


Fig. 115.

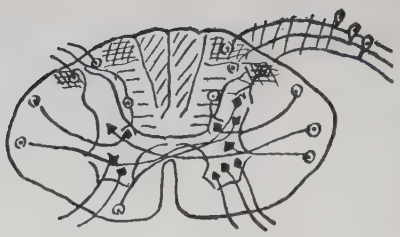


Fig. 116.

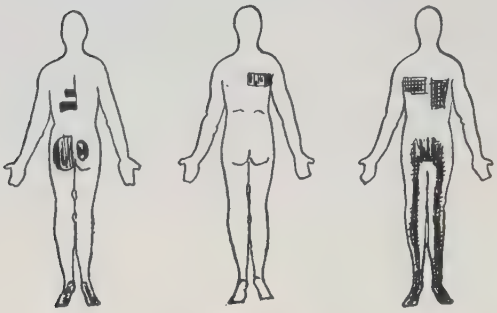


Fig. 117.

Fig. 119.

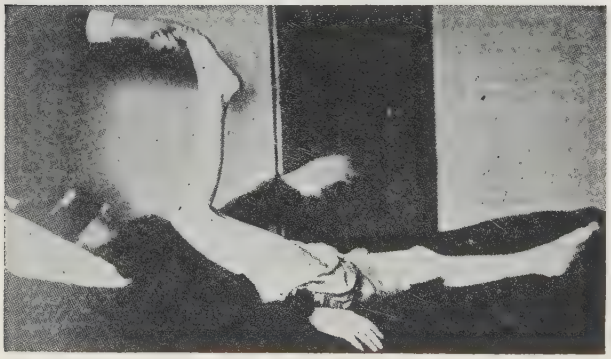


Fig. 118.

Fig. 120.

Fig. 114.—Most marked lesions in early case of locomotor ataxia. Lumbar and cervical cord of the same case. Shading shows affected area. Long sensory fibers for the leg.

Fig. 115.—Late case of locomotor ataxia. Whole posterior columns and posterolateral columns degenerated.

Fig. 116.—General plan of sensory tracts showing parts affected in locomotor ataxia. Oblique shading—Long tracts for leg affected early. Horizontal shading—Long tracts for arm affected later. (Both carry muscle sense.) Criss-cross shading in postero-lateral columns—Short tracts for pain, heat and cold irregularly affected for short distances (one or two segments) only. Disease probably starts in the nerve roots proximal to the ganglia.

Fig. 117.—Areas of anæsthesia in cases of locomotor ataxia. Compare with Fig. 96.

Fig. 118.—Extreme inco-ordination in locomotor ataxia. From instantaneous photograph while patient was trying to find her right foot with her left hand. She had no knowledge of the position of the foot when her eyes were closed and the foot was raised from the bed; but she knew the physician had hold of her great toe. (Bramwell.)

Fig. 119.—Perforating ulcer of the foot in locomotor ataxia. (Bramwell.)

Fig. 120.—Charcot's joint disease. Great synovial distension with exostoses of right knee. (Bramwell.)

Figs. 119 and 120 illustrate trophic changes in locomotor ataxia.

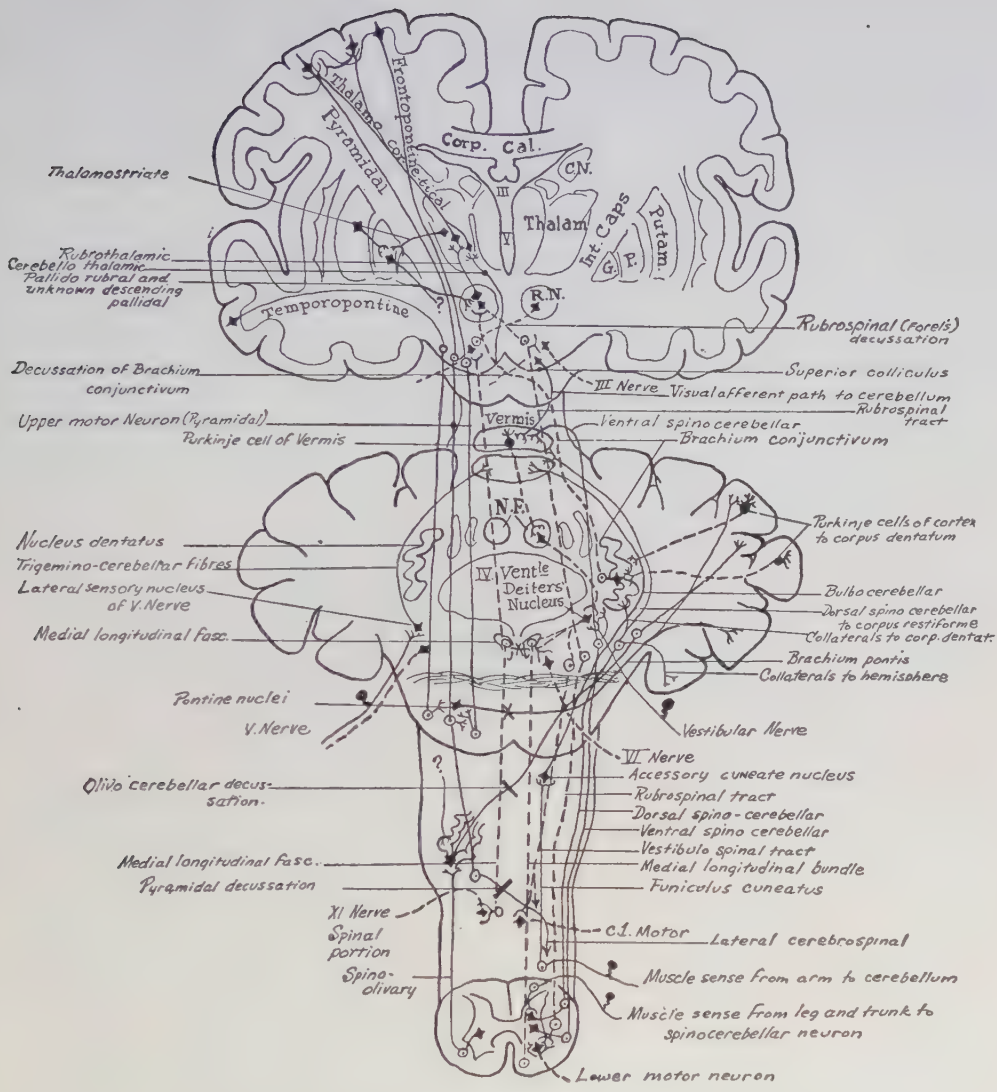


FIG. 121.—Cerebellar and vestibular afferent and efferent paths. III, V, Third ventricle; C.N., caudate nucleus; G.P., globus pallidas; R.N., red nucleus; N.F., nucleus fastigii.

For the sake of clearness the connections are drawn on one side only. For conformity the V. nerve nucleus should be on the right side but there was not room for it. Note: Cerebro-cerebellar paths are all crossed; spino-cerebellar connections are mostly ipsilateral. (W. K., 1920.)

Fig. 122.

Fig. 123.

Fig. 124.

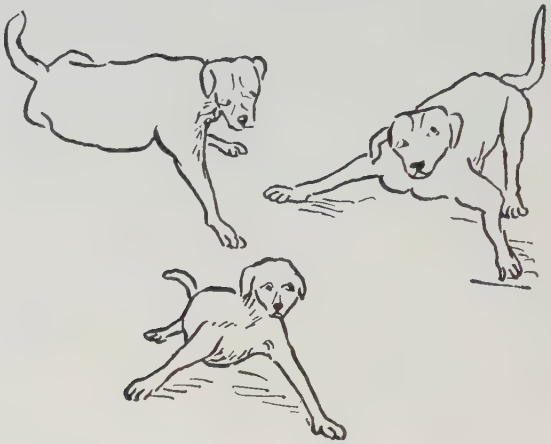


Fig. 125.

Fig. 126.

Fig. 127.

Fig. 122.—Right cerebellar hemisphere destroyed. Dog falls over on the right (homolateral) side and continues to rotate in direction in which it falls. Paralytic deviation of eyes downward and inward on the right, upward and outward on the left. (A. Thomas.)

Fig. 123.—Attitude of dog after left half of cerebellum had been destroyed. Concavity of trunk to operated side. This picture and the next taken during first few days after operation.

Fig. 124.—Same dog as Fig. 123; about to fall over to left (operated) side; left forepaw suddenly adducted. Shows inco-ordination of equilibration mechanism.

Fig. 125.—Right cerebellar hemisphere removed; right vestibular root cut. Dog learning to walk. Note skew deviation of eyes. Great abduction of right leg, difficulty in balancing. Occiput rotated to operated side. The effect is the same with hemisphere alone removed but education is easier.

Fig. 126.—After total destruction of cerebellum. Extreme abduction for forelegs.

Fig. 127.—Learning to walk after total destruction of cerebellum. Extreme abduction of forelegs. (Figures 122 to 127 from André Thomas.)

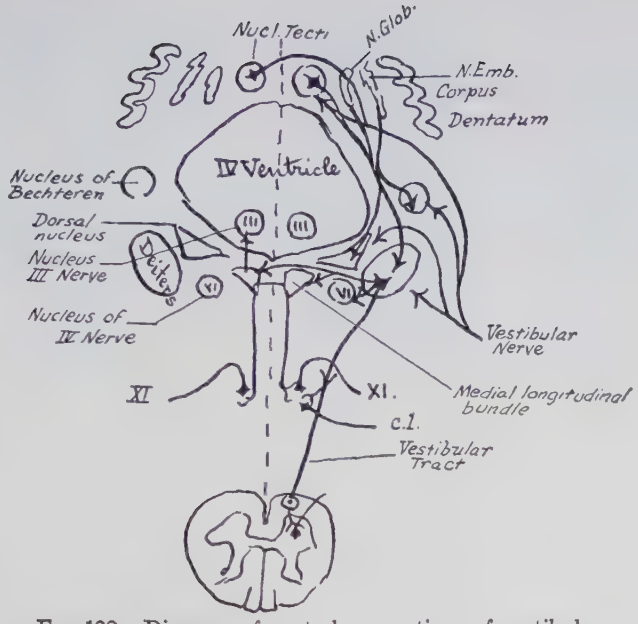


FIG. 128.—Diagram of central connections of vestibular nerve. (Modified from André Thomas.)

FIG. 128A.—Illustrating cerebellar atonia in a case of severe injury of the right lateral hemisphere of the cerebellum ten weeks after the infliction of the wound. The right thigh can be fully flexed on the trunk, the heel placed on the buttock, and the foot too much dorsiflexed with the exertion of very little power by the examining surgeon. (Gordon Holmes.)

FIG. 128B.—Illustrating cerebellar hypotonia. A case of extensive injury of the left side of the cerebellum, from a photograph taken one week after the wound. When the forearms were held vertically, the left wrist flexed under the influence of gravity much more than the right, owing to the hypotonic condition of the muscles. (Gordon Holmes, Brain, 1917.)

FIG. 128C.

FIG. 128D.

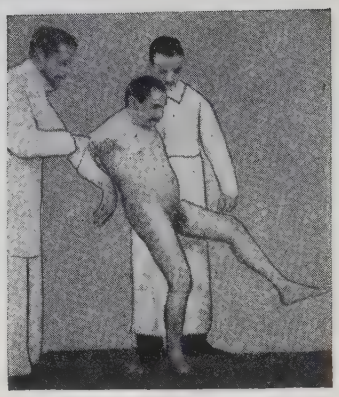


FIG. 128E.

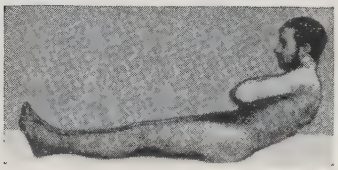


FIG. 128F.

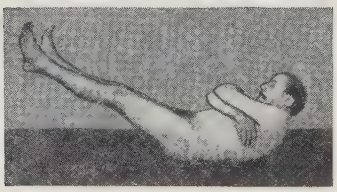


FIG. 128G.

FIG. 128H.

FIGS. 128C and D.—Illustrating cerebellar asynergia. Fig. 128C—Attitude of normal man throwing back his head and upper part of trunk. There is cerebellar synergic adjustment of the center of gravity. Fig. 128D.—Contrast this with Fig. 128C. Attitude of patient with cerebellar defect throwing back his head and upper part of trunk, showing lack of cerebellar synergic adjustment of center of gravity. (Babinski, Rev. Neurologique, 1899.)

FIG. 128E.—Showing cerebellar dys-synergia. Patient with cerebellar defect fails synergically to carry forward his trunk with his legs in walking. (Babinski.)

FIGS. 128F and G.—Illustrating cerebellar dys-synergia. Fig. 128F.—Attitude of normal man raising himself from lying to sitting position. Synergic control of legs. Fig. 128G.—Attitude of patient with cerebellar defect showing loss of cerebellar synergic control of legs when he attempts to raise himself from lying to sitting position. (After Weisenberg, U. S. Army Manual of Neuro-Surgery, 1919.)

FIG. 128H.—Method of testing cerebellar dysmetria. Patient with left-sided cerebellar defect. In performing the finger-to-nose test the patient misses the nose and hits the eye.

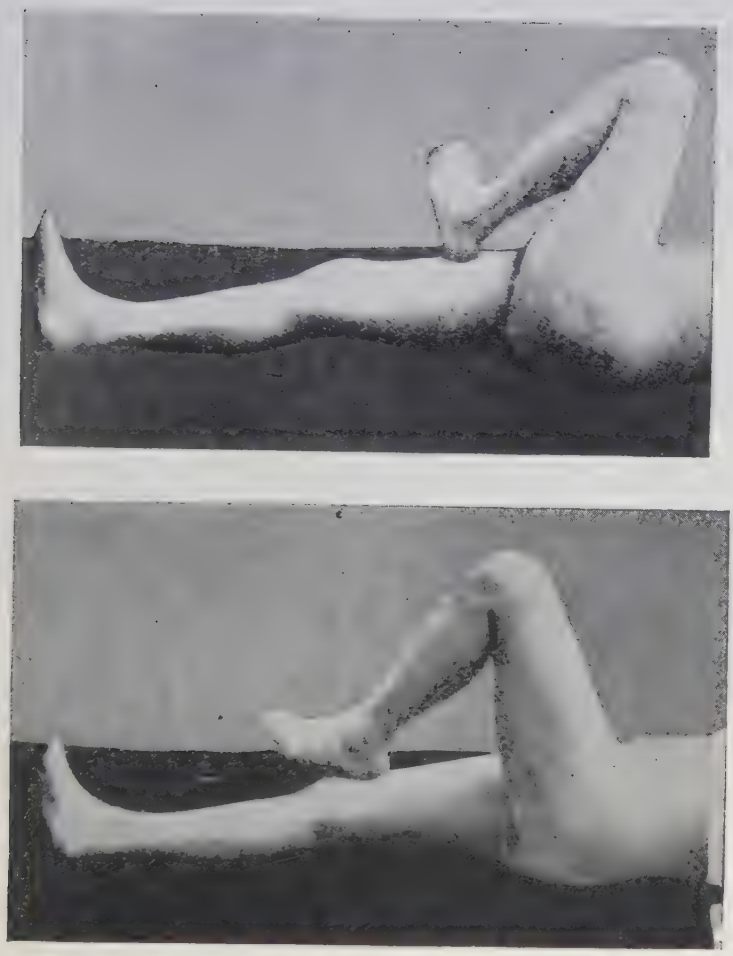


FIG. 1281.—Method of demonstrating dysmetria of lower limb in patient with cerebellar defects. In this case patient had multiple sclerosis affecting the cerebellum. When asked to place the left heel on the right knee he over-flexed his thigh, placing the heel on the opposite thigh and then sliding it down to the knee. (Dejerine Semiologie des Affections de Systeme Nerveuse.)

Fig. 129.—Illustrating cerebellar dysmetria. Patient with cerebellar atrophy, worse on left side. Patient opens his hand too much when he sets down a glass.

Fig. 130.—Softening of left restiform body. Cerebellar gait showing disturbance of equilibrium, enlarged base of support. Abduction of arm, especially left. (André Thomas.)

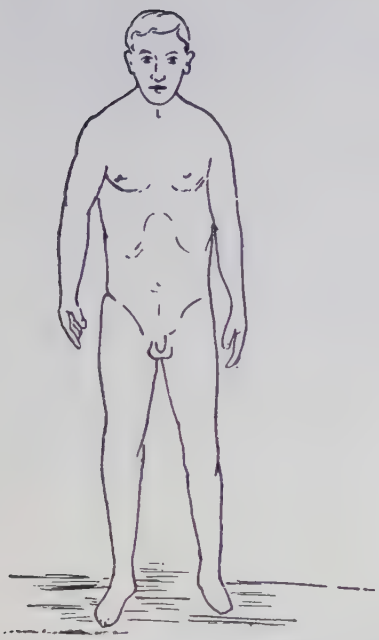


Fig. 131A.—Gait of patient with cerebellar atrophy, worse on left side. Enlarged base of support. Slow and uncertain gait. The arms do not swing synergically with the legs in walking. (André Thomas.)

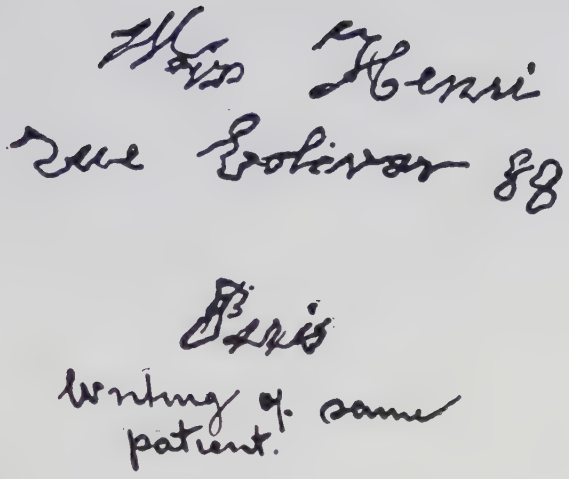


Fig. 131B.—Showing cerebellar tremor. Writing of patient in Figs. 129 and 131.

Fig. 132.—Showing adiadochokinesis in patient with left-sided cerebellar defect. In alternately pronating and supinating the forearm the left forearm overacts and lags behind the right. (After Weisenberg, U. S. Army Manual of Neuro-surgery, 1919.)

Fig. 133.—Progressive lenticular degeneration. Open-mouthed spasticity. Contractures, emaciation. Gower's case. (S. A. K. Wilson, Brain, Vol. XXXIV.)

Fig. 134.—Homen's case of progressive lenticular degeneration. Note fixed smiling expression, open mouth, contractures, emaciation. (S. A. K. Wilson, Brain, Vol. XXXIV.)

Fig. 135.—Characteristic spastic fixed smile and spasticity of hands in progressive lenticular degeneration. (S. A. K. Wilson, Brain, Vol. XXXIV.)

Fig. 136.—Progressive lenticular degeneration. Open mouth, half protruded tongue, vacant expression, wide staring eyes. Yet face is in repose. (Sawyer's case, Brain, Vol. XXXV.)

Fig 136A.—Progressive degeneration of globus pallidus. Aged 31; 16 years after onset of disease. Note fixed smile, bright intelligent eyes, and Parkinsonian deformities of hand and fingers. (Ramsay Hunt, Brain, 1917.)

Fig. 136b.—Progressive atrophy of the globus pallidus at the age of 17, two years after the onset of the disease. Note the attitude, position of hands and fingers and the well-defined contour of the muscles (rigidity). (Ramsay Hunt, Brain, 1917.)

Fig. 136c.

Fig. 136c.—Torsion spasm in postencephalitic striatal degeneration. A, spasm in sitting position; B, lying in bed; C, Convulsions at times turned him face downward. (Woods and Pendleton. Trans. Am. Neurol. Assoc., 1924.)

Fig. 137.

Fig. 137.—Paralysis Agitans. Chronic degeneration of the pallidal system. Attitude of the head, trunk and hands. Patient, aged 54; disease commenced at 40. (Dejerine's Semiologie.)

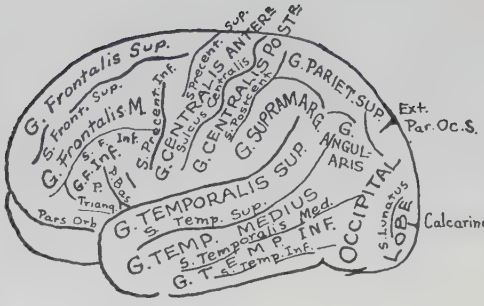


Fig. 138.—Fissures and convolutions of lateral surface of left hemisphere.

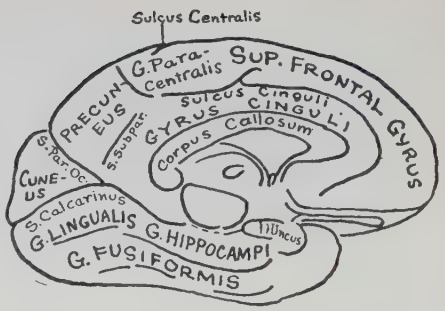


Fig. 139.—Fissures and convolutions of inner surface of left hemisphere.

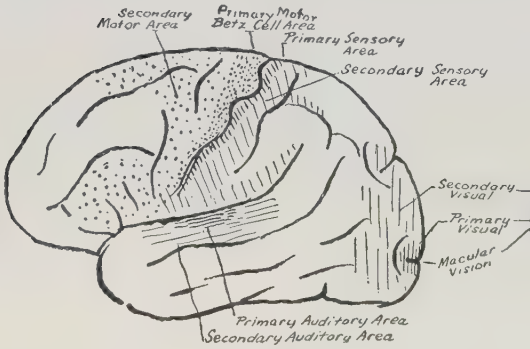


Fig. 140.

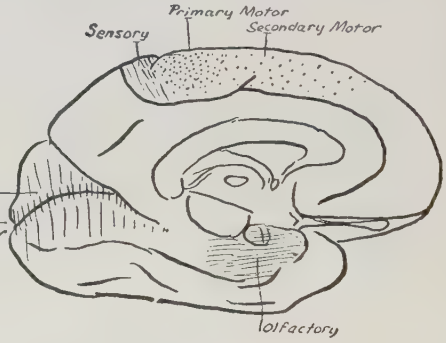


Fig. 141.

Figs. 140 and 141.—Campbell's cell areas as applied to the more important motor and sensory regions of the cortex.

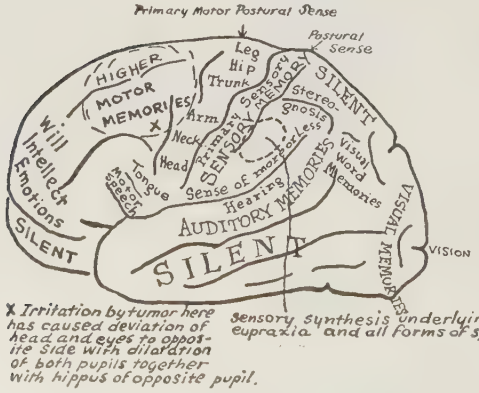


Fig. 142.—Localizing areas in left hemisphere. Lateral surface. X, head and eyes.

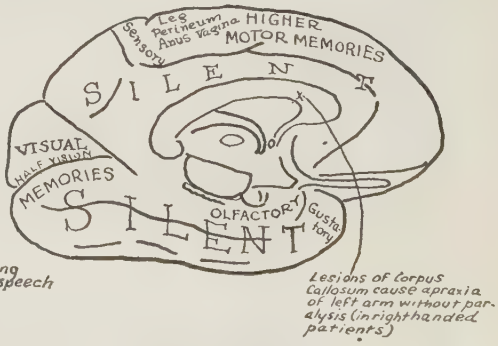


Fig. 143.—Localizing areas in medial surface of left hemisphere.

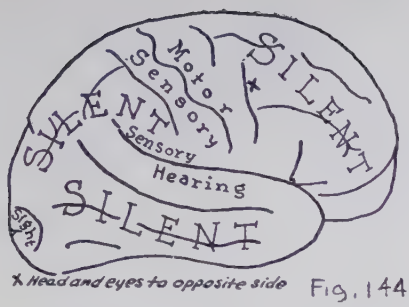


FIG. 144.—Localizing areas, right hemisphere. Contrast with Fig. 142.

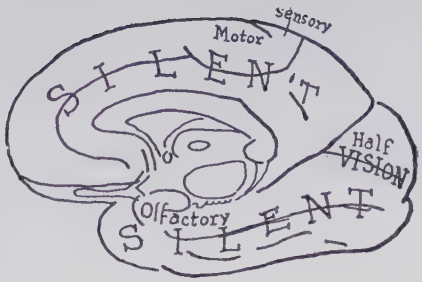


FIG. 145.—Localizing areas, medial side of right hemisphere. Contrast with Fig. 143.

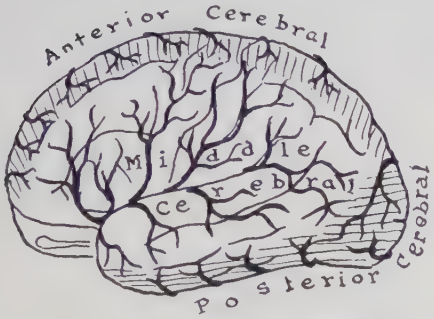


FIG. 146.—Arteries of the outer surface of the hemisphere. Middle cerebral supplies motor area, except part of leg area. It also supplies the auditory area. Posterior cerebral supplies visual area. Shading indicates the three areas: unshaded area, middle cerebral; vertical shading, anterior cerebral; horizontal shading, posterior cerebral.

FIG. 147.—Arteries of the inner surface of the hemisphere. Anterior cerebral artery supplies corpus callosum and motor and sensory area for part of leg and perineum. Posterior cerebral supplies visual and olfactory area.

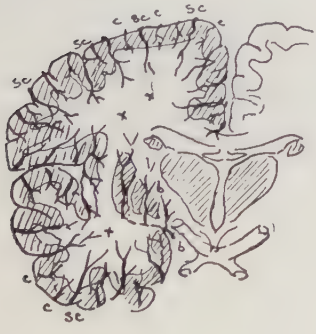


FIG. 148.—Shows C, cortical arteries; Sc, subcortical arteries; b, central arteries; x, x, ischaemic area, area of cerebral softening.

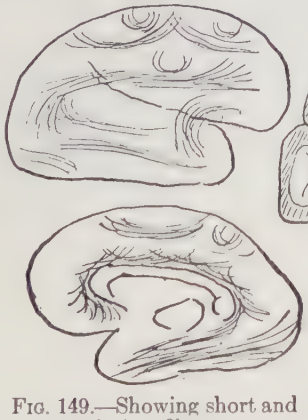


FIG. 149.—Showing short and long association fibers.

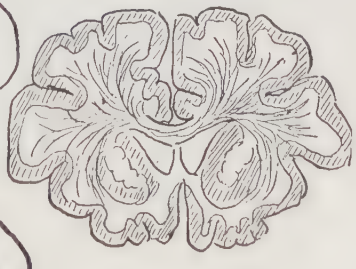


FIG. 150.—The corpus callosum is the great transverse commissure uniting similar (?) areas of each hemisphere to its fellow.

FIGURES 151 to 166 ILLUSTRATE LOWER MOTOR NEURON PARALYSIS FROM VARIOUS CAUSES

FIG. 151.—Drop wrist from paralysis of the radical (musculospiral) nerve due to sleeping with arm over chair.

FIG. 152.—Paralysis, wasting, late contractures and trophic changes, especially in the nails, 44 years after section of the median and ulnar nerves at the wrist.

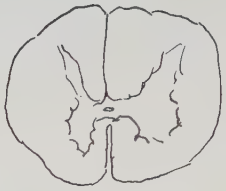


FIG. 153.—Cervical spinal cord in an old case of infantile paralysis. Left anterior gray column especially is shrunken. (Church and Peterson.)

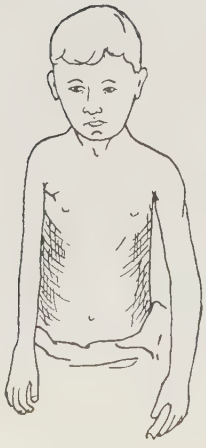


FIG. 154.—Atrophy of right upper limb and thorax after acute anterior poliomyelitis. (Church and Petersen.)

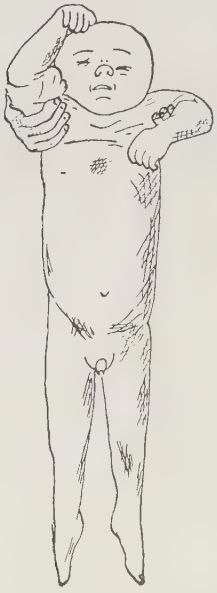


FIG. 155.—Severe acute anterior poliomyelitis affecting lumbar enlargement. Legs hang like useless appendages. From instantaneous photograph while child is struggling. (Byrom Bramwell.)

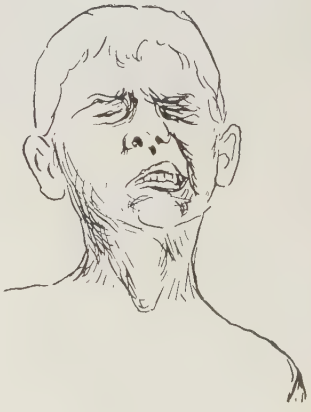


FIG. 156.—Bulbopontine form of acute anterior poliomyelencephalitis. Paralysis of right side of face with flaccid paralysis of left shoulder. Patient is closing his eyes forcibly. (Batten, Brain, 1916.)

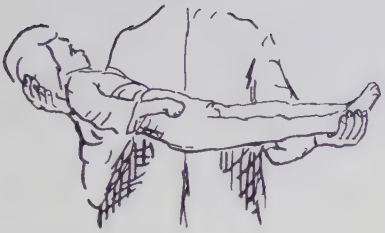


FIG. 157.—Pseudorigidity in poliomyelitis. Legs rigid due to unbalanced action of gluteal muscles. Flexors of thighs paralyzed. (Batten, Brain, 1916.)

FIG. 158.—Bulging of abdominal wall due to poliomyelitis. (Batten, Brain, 1916.)

FIG. 159.—Poliomyelitis affecting gluteal muscles. Bilateral contraction of hip joint flexors. (Brain, 1916.)

FIG. 160.—Anterior gray column from a case of progressive muscular atrophy. Lower motor neuron type. (Bramwell.)

FIG. 161.—Normal gray column for comparison. Note shrinking and absence of L. M. N. cells in diseased cord.

FIG. 162.—Progressive spinal muscular atrophy. Forearms and hands most affected. Pectorals and chest muscles also involved. Cord as in Fig. 160. (Bramwell.)

FIG. 163.—Lower motor neuron paralysis of both arms and back; patient 54 years; disease began at the age of 24. In addition to muscular wasting, there is loss of pain, heat and cold for both arms. Disease: syringomyelia. Cord as in Fig. 164. Anterior gray columns and posterior spino-thalamic neurons in gray matter destroyed. (Dejerine.)

FIG. 164.—Cervical cord in syringomyelia which would cause symptoms like Fig. 163.

FIG. 165.—Alcoholic peripheral neuritis. Wrist drop and foot drop. Cirrhosis of the liver. Ascites. (Monographic Medicine, Barker.)

FIG. 166.—Birth palsy from traction in axilla. 8th cervical and 1st thoracic nerves affected. (Church and Peterson.)

FIGURES 167 to 172 ILLUSTRATE TYPICAL LESIONS CAUSING UPPER MOTOR NEURON PARALYSIS

FIG. 167.—Sites of cortical lesions causing typical cortical upper motor neuron paralysis. Dotted area—cortical lesion here causes monoplegia of right leg; horizontal shading—monoplegia right arm; oblique shading—right facial monoplegia; vertical shading—motor aphasia.

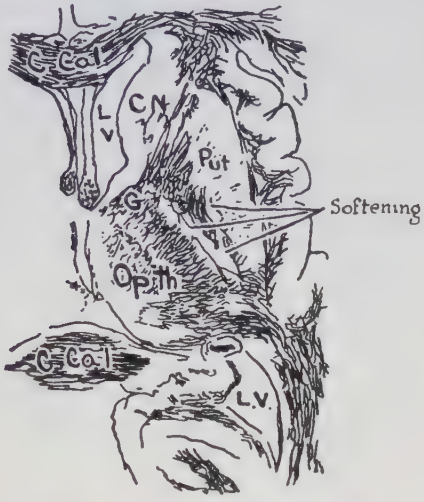


FIG. 168A.—Patient, Wm. T. Softening in right internal capsule. Pal-Weigert stain. Hemiplegia dating back one year. C. Cal., corpus callosum; C. N., caudate nucleus; Put., putamen; Op. th., optic thalamus; L. V., lateral ventricle; G., genu of internal capsule.

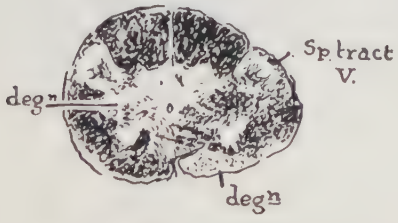


FIG. 168B.—Patient, Wm. T. Pyramidal decussation. Right pyramidal tract shows degeneration. Pal-Weigert stain.

Fig. 169A.—Softening and degeneration of the left side of pons; case B.

Fig. 169B.—Case B. Showing descending degeneration in left pyramid. (Unfortunately the slide was inverted.)

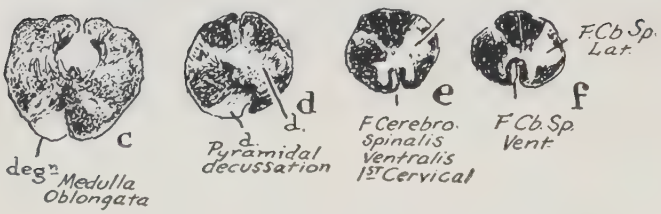


Fig. 169C.—Softening of pons causing hemiplegia with degenerations. (Pal-Weigert.)

Fig. 171.—Geo. W. A., aet., 36. Combined sclerosis. Seven months before death pains in feet, ankles, knees and lower abdominal muscles; two years before death burning tingling pain under both feet; six months before death symptoms of spastic paraplegia of legs. Acute symptoms before death. Softening of 8th, 9th and 10th thoracic segments hastened death. Drawing shows lumbar cord. Patient had a secondary anæmia. (Pal-Weigert.)

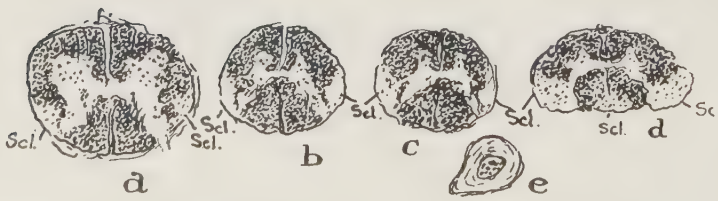


Fig. 170.—Voss, aet. 70. Slowly progressive lateral sclerosis confined to spinal cord. Pyramidal decussation showed no sclerosis. Arteries (e), especially in thoracic and cervical cord, show marked hyaline thickening. Chief symptom, weakness in legs for 30 years. a, Lumber enlargement; b, about mid thoracic; c, 1st lumbar; d, 7th cervical; e, artery from gray column, high power. The cells of the anterior gray columns are normal except for some recent degenerative changes. Note that contrary to custom in these drawup the dorsal surface of the sections in Fig. 170 is toward you.

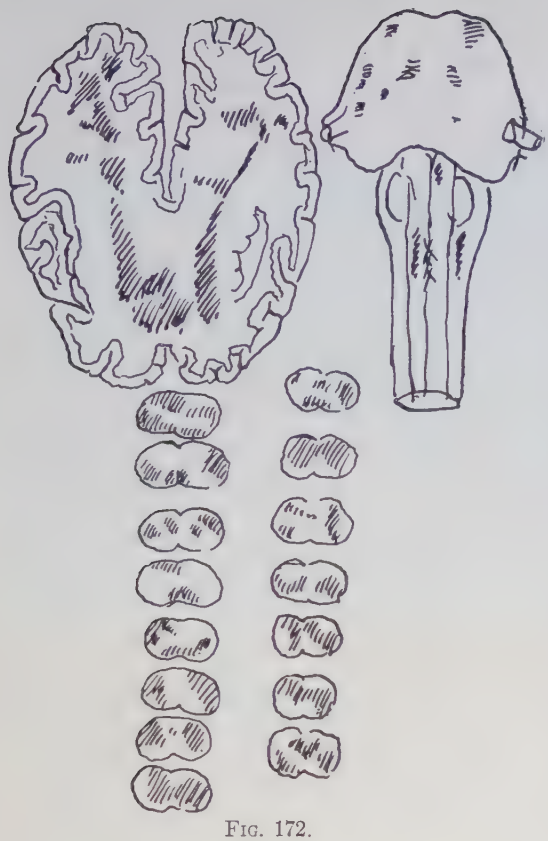


Fig. 172.

Fig. 172.—Multiple cerebrospinal sclerosis. Sections at various levels of the same case. Shaded areas show sclerosis. (Charcot.)

Fig. 173.—Lesions causing motor apraxia in right-handed subject.

1, Lesion of posterior ends of superior and middle left frontal gyri interferes with patient's educated motor memories. Spontaneous movements perfect; volitional purposive movements imperfect with both hands.

2, Subcortical lesion in left hemisphere destroying connection of superior and middle frontal with Betz cell area. Right motor apraxia without paralysis.

3, 4, 5, Lesions interrupting connections between area of higher motor memories and Betz cell area in right anterior central gyrus, cause left-handed apraxia without any paralysis of either hand.

6, Lesion causing right-handed paralysis with left-handed apraxia but without left-handed paralysis.

7, Lesion causing right hemiplegia; no apraxia of left hand.

8, Lesion of left supramarginal gyrus, causing complex aphasia with bilateral apraxia. See Figs. 173A, B, C.

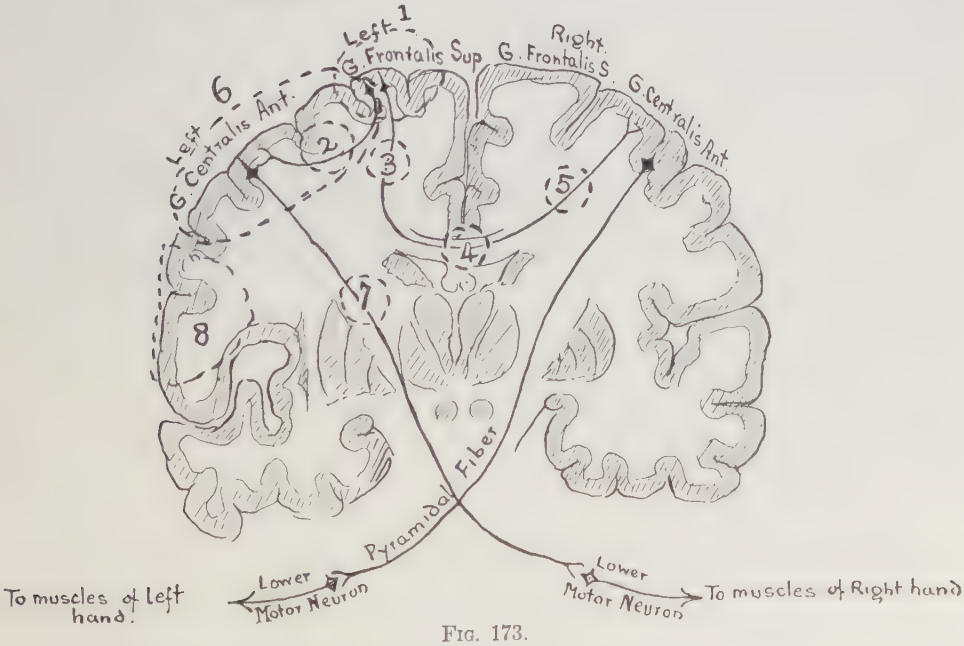


Fig. 173.

FIG. 173A.

FIG. 173B.

FIG. 173C.

FIGS. 173A, B, C.—Illustrating apraxia. See Fig. 173 for site of lesion. Case in Cushing's Clinic (Bremer, Archives of Neurol. and Psych., 1921). Endothelioma pressing on left supramarginal gyrus.

Figs. 173A and B.—Degrees of perplexity of patient after long unsuccessful attempts to light the candle. He had previously been unable to light his cigarette which was lit for him.

Fig. 173C.—Patient endeavoring to strike the unlighted match on the unlighted candle.

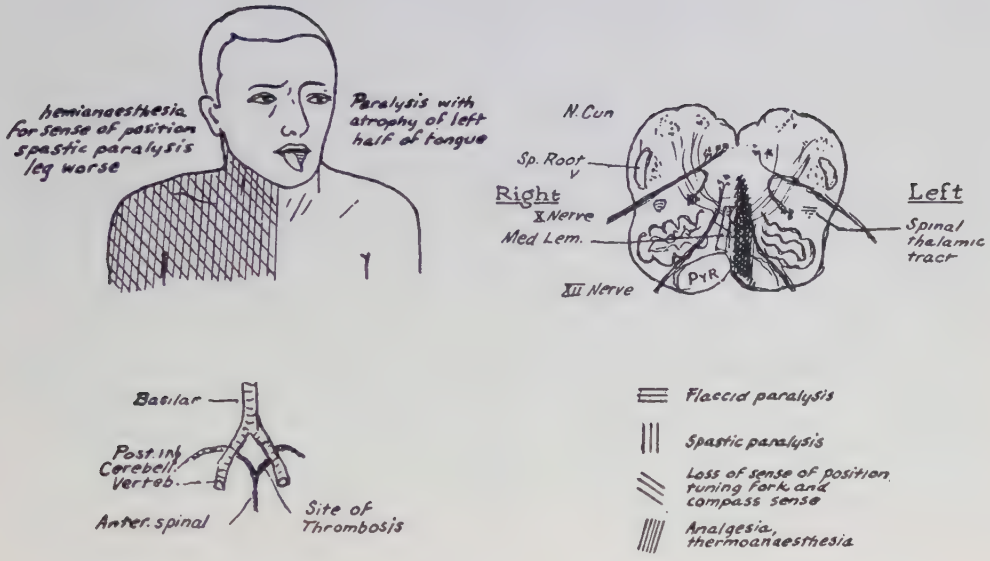


Fig. 174.—Antero-internal bulbar syndrome. Thrombosis of left anterior spinal artery.

Destroys left twelfth nerve root, part of left pyramidal tract, left medial lemniscus.

Destroys left XII Nerve, hence: Left flaccid paralysis of tongue with hemiatrophy and reaction of degeneration.

Interrupts left medial lemniscus, hence: Right-sided loss of sense of position, tuning fork sense and weight, over right half of body and limbs.

Partial destruction of left pyramid, hence: Spastic paralysis of right side of body, incomplete, leg suffering most.

No loss of pain, heat or cold as spinal thalamic tract is uninjured.

Note: Tactile discrimination does not travel upward in the medial lemniscus. (Adapted from Dejerine's Sémiologie, Fig. 49.)

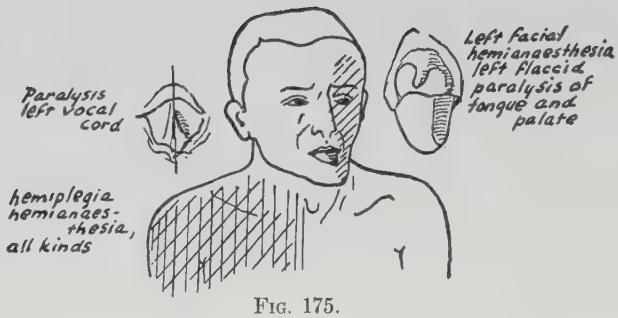

Paralysis left Vocal cord
Left facial hemianaesthesia left flaccid paralysis of tongue and palate
hemiplegia hemianaesthesia, all kinds

FIG. 175.

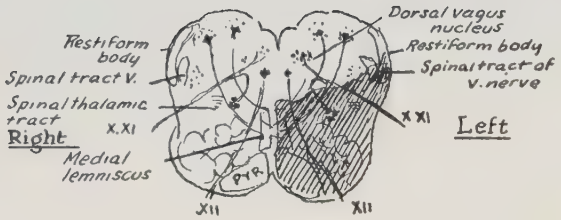


FIG. 175.

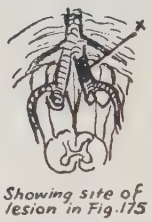


FIG. 175[1].

FIG. 175.—Antero-internal and retro-olivary bulbar syndrome.

Thrombosis of left vertebral artery in front of posterior inferior cerebellar branch (Fig. 175[1]).

Causing: *Homolateral symptoms.*

Left XII Nerve, hence: Flaccid paralysis left half of tongue with wasting and reaction of degeneration.

Left accessory nerve, hence: Paralysis of left vocal cord, left half of palate and uvula. Uvula deflected to sound side.

Left spinal tract of V Nerve, hence: Hemianæsthesia (partial) of left side of face, all forms of sensation, especially heat and cold.

Homolateral cerebellar symptoms from lesion of corpus restiforme and posterior spino-cerebellar tract; left hemiasyngeria, hemiataxia.

Homolateral drooping of eyelid and contraction of pupil from destruction of autonomic center in oblongata.

Heterolateral tract symptoms:

Left pyramidal tract, hence: Spastic paralysis of right arm and leg.

Left medial lemniscus and adjoining longitudinal fibers, hence: Right hemianæsthesia for sense of passive position, weight, compass and tuning fork sense.

Left spinothalamic tract: Right hemianalgesia, hemi-thermoanæsthesia.

(Adapted from Dejerine's Séminologie, Fig. 50.)

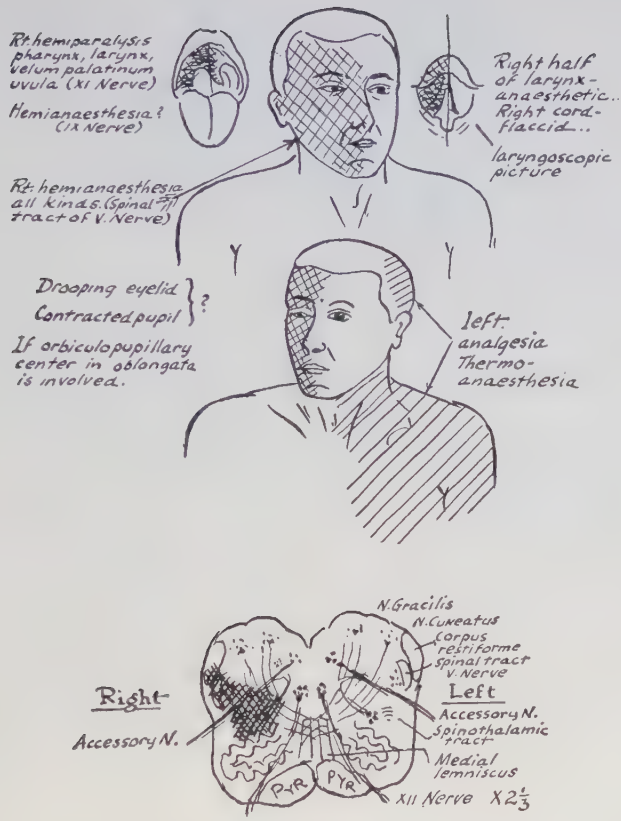


FIG. 176.—Retro-olivary bulbar syndrome.

Lesion of posterior inferior cerebellar artery.

Homolateral symptoms:

Right vague and accessory roots, hence: Right hemiparalysis and (?) hemianæsthesia of palate and larynx; uvula drawn to left; palate flaccid; right vocal cord flaccid.

Right spinal tract of V. Nerve, hence: Right hemianæsthesia of face; all kinds of sensation, especially pain, heat and cold.

Right restiform body, hence: Right cerebellar ataxia; asynergy.

Perhaps involvement of:

Sympathetic center, hence: Drooping eyelid; contracted pupil.

Heterolateral symptoms:

Destruction of right spinal thalamic tract, hence: Left hemianæsthesia for heat, cold and pain below the V. Nerve.

Pyramid and medial lemniscus escape, hence: No crossed hemiplegia, no loss of muscle-tendon-joint, position and bone sense.

(Dejerine, Fig. 60.)

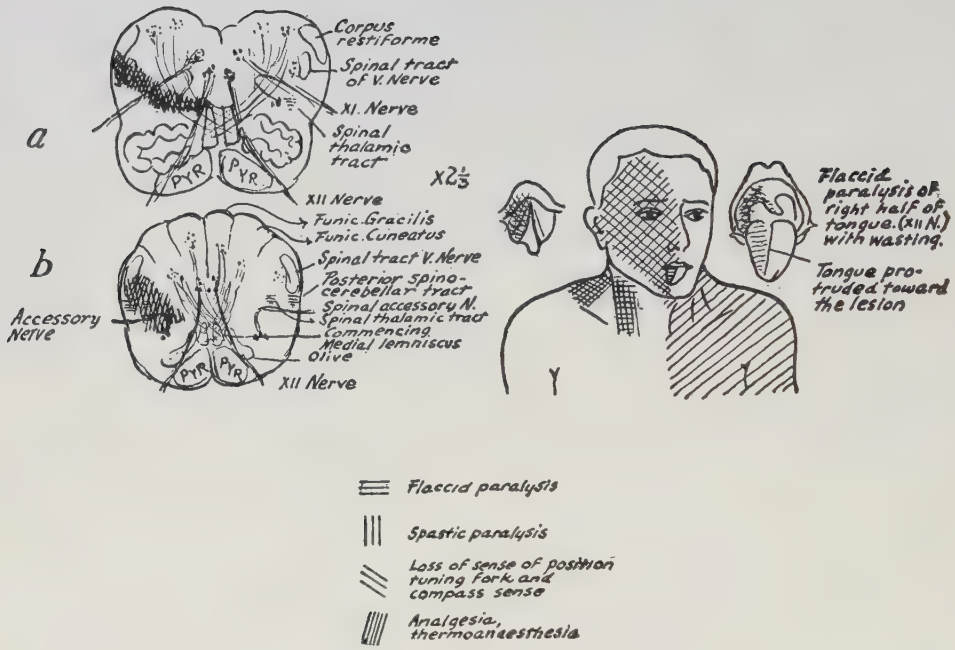


FIG. 177.—Another case of retro-olivary bulbar syndrome.

In (a) the palpebral pupillary center escapes but XII root is involved.

In (b) which shows the same lesion lower down, the roots of the XI Nerve to the trapezius and sternomastoid are interrupted.

Symptoms same as last; but without the eye symptoms and with added flaccid paralysis of right half of tongue and with paresis of trapezius and sternomastoid (they are still supplied by cervical nerves). (Dejerine, Fig. 60.)

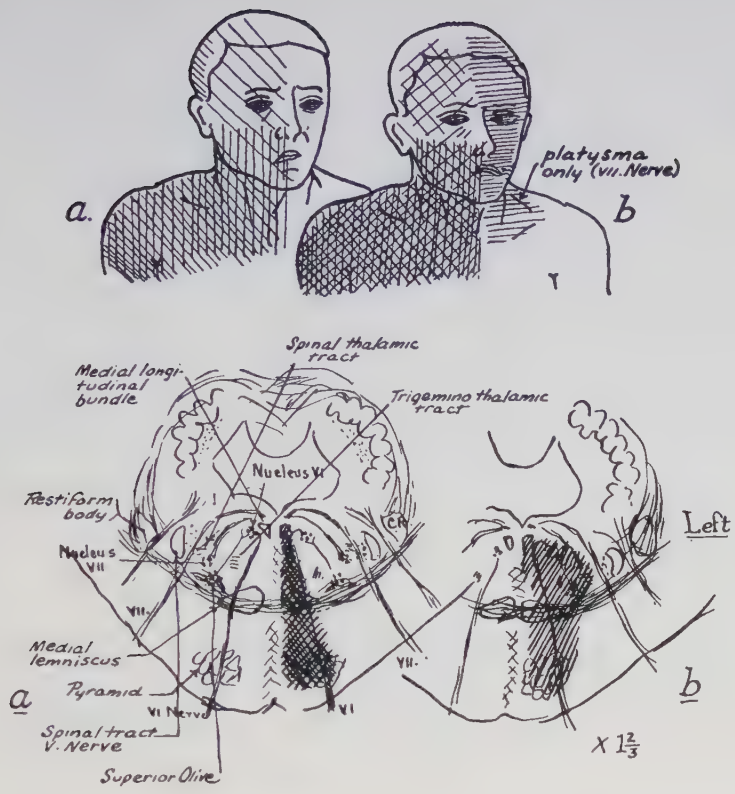


FIG. 178.—Dorsoventral lower pontine syndrome.

In (a) the left VII Nerve and spinal thalamic tract have escaped; in (b) both are involved.

Thrombus of posterior end of basilar artery especially affecting the left pontine branches.

In (a):

Homolateral symptoms:

Involvement of left VI Nerve, hence: Paralysis of left lateral rectus; eye turned inward by unopposed medial rectus.

Heterolateral symptoms:

Interruption of left medial longitudinal fasciculus, hence: Right eye cannot be turned to left side except weakly for convergence.

Interruption of left pyramidal tract, hence: Spastic paralysis of right side of body, perhaps of lower face; paresis of right half of tongue.

Interruption of left medial lemniscus, hence: Loss of tuning fork sense; sense of passive position and compass sense (?) over right side of body and limbs; face less affected.

In (b) there is in addition:

Interruption of left VII Nerve, hence: Paralysis of whole left face and platysma. Eye cannot be closed, lips sag, facial grooves lost, mouth and chin drawn to right.

Interruption of left spinothalamic tract and left trigemino-thalamic tract, hence: Right hemithermoanæsthesia and hemianalgesia in face, trunk and limbs; less in face. (Djerine.)

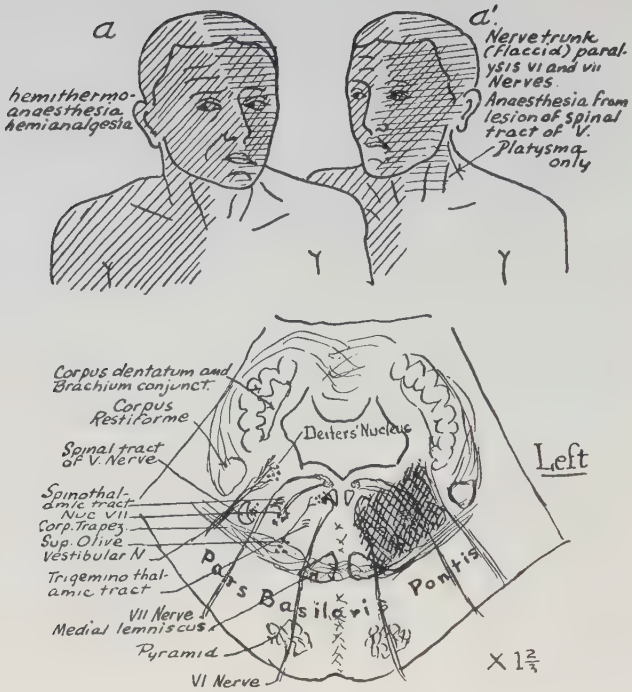


Fig. 179.—Dorsal pontile syndrome.

Case of hemorrhage, into dorsolateral area of pars tegmentalis pontis. Destroys Deiters' nucleus, VII nucleus and nerve, VI Nerve, superior olive, spinal tract of V Nerve, spinal thalamic and trigeminothalamic tracts. The medial lemniscus, medial longitudinal bundle, VI nucleus (there is a slight error in the diagram which should not include this nucleus), corpus restiforme, and basilar portion of pons *escape.*

Homolateral symptoms:

Interruption of VI Nerve, hence: Paralysis of left lateral rectus. Eye turned inward by unopposed medial rectus.

Destruction of left Deiters' nucleus, hence: Destruction of vestibular mechanism for turning both eyes to left. Therefore, right eye turned to right, as the feeble action of medial rectus is overcome by lateral rectus. (Would destruction of superior olive have a similar effect?)

This is properly a heterolateral symptom, see below.

Destruction of left VII Nerve and nucleus, hence: Flaccid paralysis of left face muscles and platysma. Left eye cannot be closed. Angle of mouth droops, mouth and chin drawn to right, wrinkles lost.

Destruction of left spinal tract of V Nerve, hence: Anæsthesia, especially thermo-anæsthesia and analgesia of left face (not complete).

Heterolateral symptoms:

Destruction of vestibular (Deiters' nucleus), hence: Right eye cannot be rotated to left. See above.

Destruction of spinothalamic and trigeminothalamic tracts, hence: Right hemithermo-anæsthesia and hemianalgesia over face and body and limbs; face probably less as upper part of tract for face escapes.

No loss of muscle sense or hemiplegia on right side.

(Dejerine, Fig. 58.)

FIG. 180.—Anterior mesencephalic syndrome (Weber's).

(a)

Homolateral symptoms:

Left III Nerve, hence: Left ptosis. Eye turned out by unopposed lateral rectus.

Crossed symptoms:

Middle of left basis mesencephali, hence: Spastic paralysis of right side of body including face and tongue.

Note in (a) the pyramidal fibers to the right fifth accessory and sixth nerves have escaped. The tongue and upper face are only paretic owing to homolateral pyramidal supply.

(b)

Same as (a) but the lesion is more extensive and pyramidal fibers to the accessory, fifth and sixth nuclei are involved so that there is paresis of the soft palate, uvula, sternomastoid, larynx and masticatory muscles. In addition to the symptoms of (a) there are, therefore, the following *crossed* symptoms:

Pyramidal fibers to right V nucleus, hence: Paresis of right muscles of mastication.

Pyramidal fibers to right VI nucleus, hence: Right eye turned in by unopposed right medial rectus.

Pyramidal fibers to accessory nucleus, hence: Paresis of right half of velum palatini and larynx and right sternomastoid.

(Dejerine, Fig. 53.)

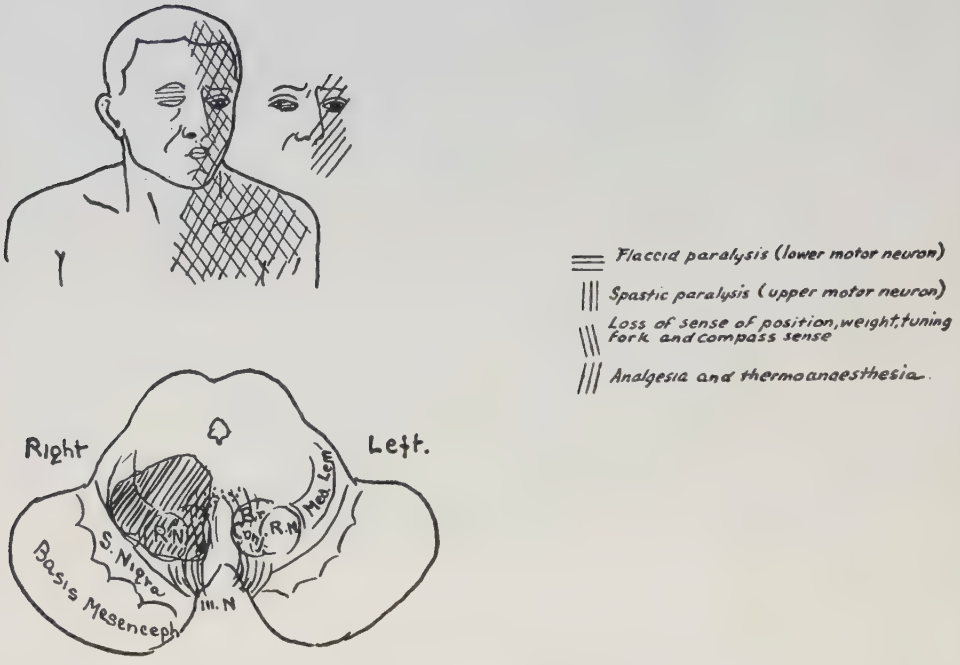


FIG. 181.—Tegmental mesencephalic syndrome (Benedict).

Homolateral symptoms:

Lesion of right III Nerve, hence: Ptosis of right eyelid. Mydraisis of right pupil. Right eye turned out by unopposed action of lateral rectus.

Crossed symptoms:

Lesion of right medial lemniscus and right spinothalamic and trigeminothalamic tracts, hence: Left hemianæsthesia for all forms of sensation; left hemiataxia of central type.

Lesion of right red nucleus and brachium conjunctivum above the decussation, hence: Left tremor; choreio-athetoid movements. Other cerebellar symptoms.

Note the pupillary and ciliary fibers of the III Nerve sometimes escape.

(Dejerine, Fig. 54.)

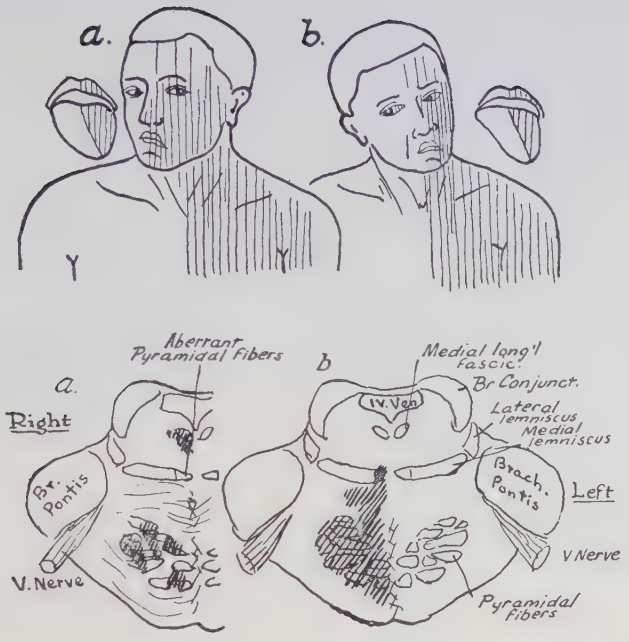


FIG. 182.—Upper pontine syndrome.

In (a) there is also lesion of right medial longitudinal bundle.

(a)

Foville's syndrome; irregular lesions of pyramidal bundles; the fibers to the left VI and left XI nuclei escape.

There is spastic paralysis of the left lower face and left extremities; weakness of the left half of the tongue and upper face.

The left external rectus and left sternomastoid escape.

The lesion of the right medial longitudinal bundle causes inability to turn the eyes to the right, due to loss of vestibular connection.

(b)

Pyramidal fibers to left VI and XI nuclei involved in medial end of medial lemniscus (Dejerine's aberrant pyramidal fibers).

Spastic paralysis of left side of body (limbs) and lower face.

Paralysis of left lateral rectus. Left eye turned in by unopposed action of medial rectus.

Right eye weak for conjugate deviation to left, because of loss of pyramidal control.

Left sternomastoid paralyzed and head turned to left by unopposed right sternomastoid.

No sensory loss. Rigidity of pure pyramidal type.

(Dejerine, Fig. 57.)

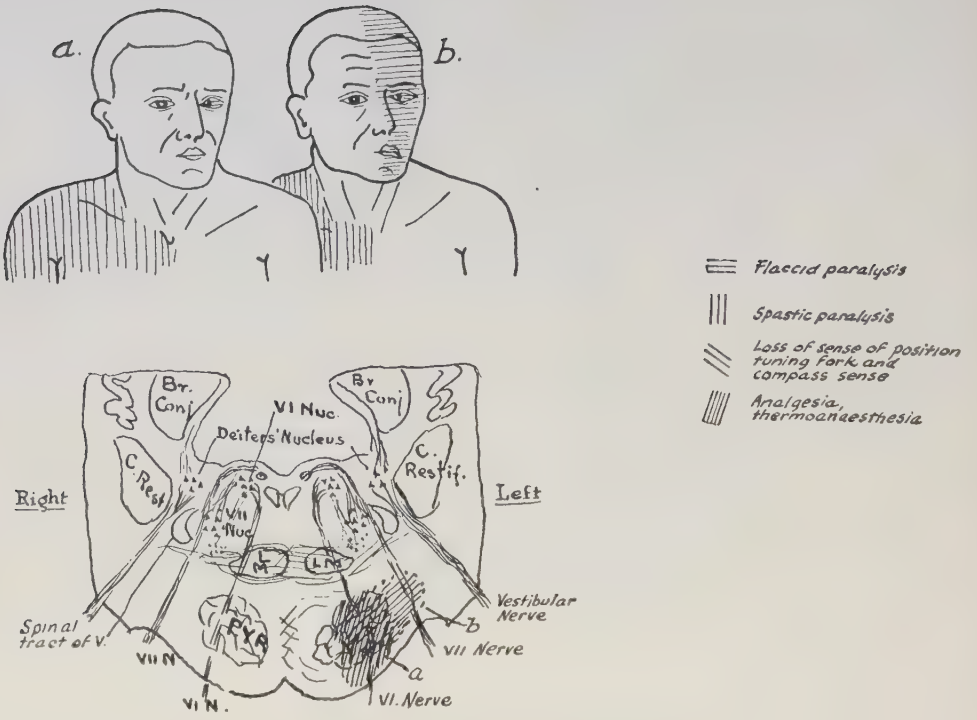


FIG. 183.—Lower pontine syndrome: ventral lesions.

(a)

Destruction of left pyramidal bundles (the aberrant fibers to XI and XII in the medial lemniscus escape), together with the emerging fibers of the VI Nerve.

Homolateral symptoms:

Interruption of left VI Nerve roots, hence: Paralysis of left lateral rectus with internal strabismus of left eye.

Crossed symptoms:

Interruption of left pyramidal tract above the pyramidal decussation; the aberrant fibers escape, hence: Hemiplegia of right half of body below the face. Face, tongue, larynx, pharynx, and sternomastoid escape. Rigidity of pure pyramidal type.

(b)

Same as (a) plus destruction of emerging VII Nerve. Syndrome of Millard Gubler.

Symptoms:

Same as (a) plus L. M. N. paralysis of left VII Nerve; flaccid paralysis of whole left face, including frontalis, orbicularis oculi and platysma.

No sensory loss.

(Dejerine, Fig. 55.)

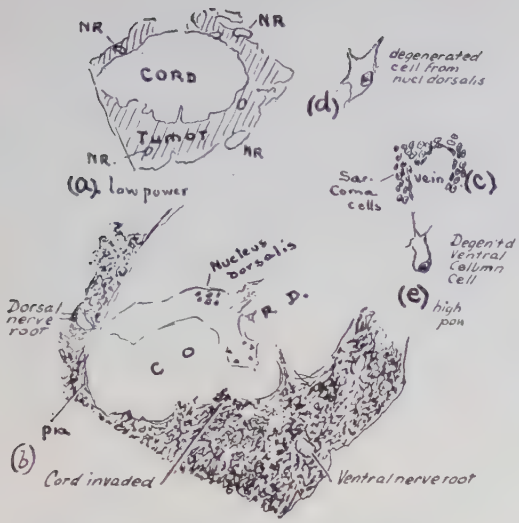


Fig. 184.—Sarcomatosis of Leptomeninges of brain and cord. (Path. Lab., U. of T., W. K.)

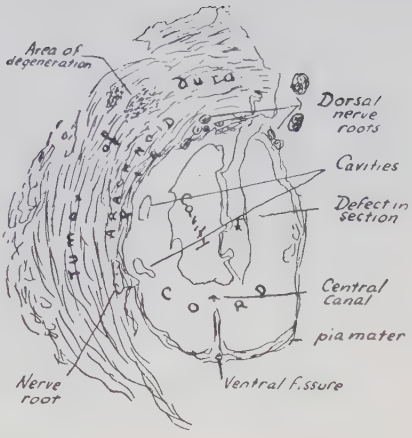


Fig. 185.—Hypertrophic pachymeningitis, syphilitic, causing complete interruption of spinal cord at 5th thoracic segment. (For degenerations, see Fig. 82.) (Anat. Lab., U. of Tex., W. K.)

Fig. 186.—Cord: Amyotrophic lateral sclerosis, seventh cervical segment. Almost complete loss of L. M. N. cells. Shading indicates area of sclerosis. (U. of Tex., Autopsy 1383, W. K.) Normal cord, sixth cervical, for comparison of L. M. N. cells. (W. K.)

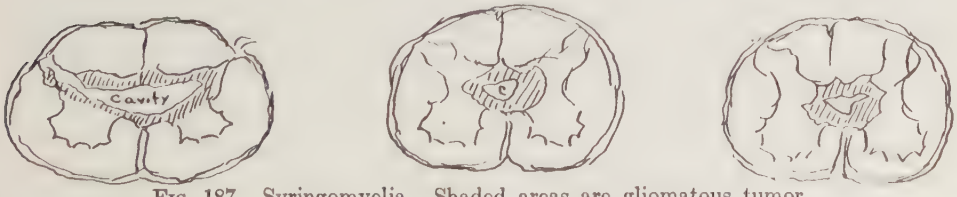


Fig. 187.—Syringomyelia. Shaded areas are gliomatous tumor. (Bruhl, Church and Peterson.)

FIG. 188.—Syringomyelic cavitation destroying gray matter. (Brissaud, Church and Peterson.)

FIG. 189.—Friedreich's ataxia. Seventh cervical segment.

FIG. 190A and B.—Tabes dorsalis, advanced case. Myelin stain. a, Thoracic cord; b, Lumbar enlargement. (Anat. Lab., U. of T., W. K.)

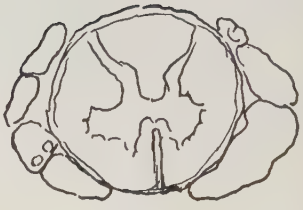


FIG. 191.—Syphilitic leptomeningitis destroying nerve roots. (Starr.)

FIG. 192.—Leprosy. a, Median nerve showing advanced fibrosis. b, Second thoracic segment of spinal cord, showing secondary posterior sclerosis. Myelin stain. (Anat. Lab., U. of T., W. K.)

Fig. 193.

Fig. 194.

Fig. 195.

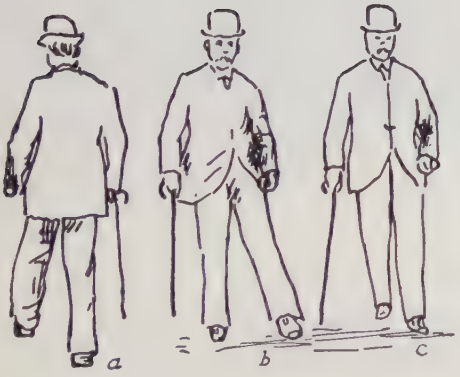


Fig. 196.

Fig. 197.

Fig. 198.

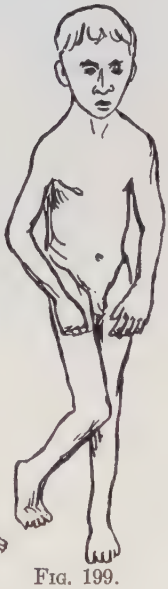


Fig. 199.

Fig. 193.—Left facio-brachial monoplegia from embolism. Photograph taken on 15th day after onset. Upper face weak only (left eye more open than right from paresis of orbicularis oculi). Lower face completely paralyzed. (Dejerine.)

Fig. 194.—Ordinary attitude of upper limb in hemiplegia. (Dejerine.)

Fig. 195.—Hemiplegia for 8 years. Late deformity with atrophy. Patient aet. 67. (Dejerine.)

Fig. 196.—Hemiplegic gait. (a) and (b), advancing hemiplegic leg in circle from hip; (c), bearing weight on paretic leg and cane while advancing sound leg. (Church and Peterson.)

Fig. 197.—Marked disuse atrophy of left arm in hemiplegia; patient aet. 43; 9 years after onset. Note attitude of arm.

Fig. 198.—Infantile cerebral monoplegia. (Jelliffe and White.)

Fig. 199.—Child of 9 years with congenital double hemiplegia. Legs worse; arms improving. (Dejerine.)

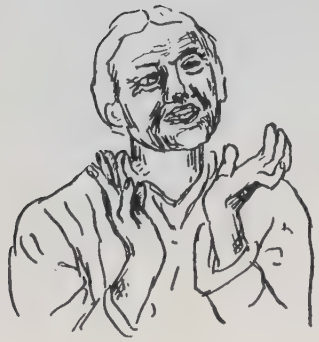


Fig. 200.

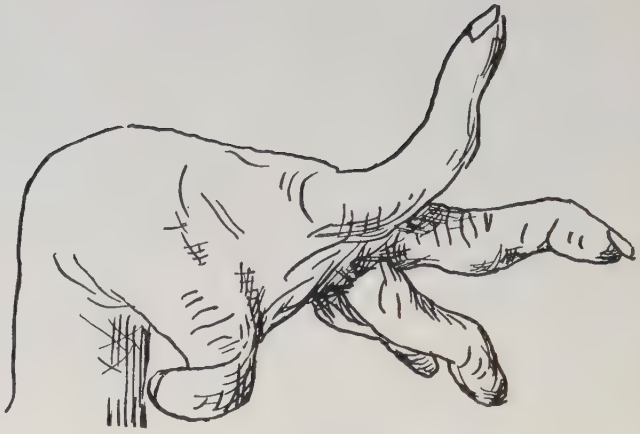


Fig. 201.

Fig. 202.

Fig. 203.

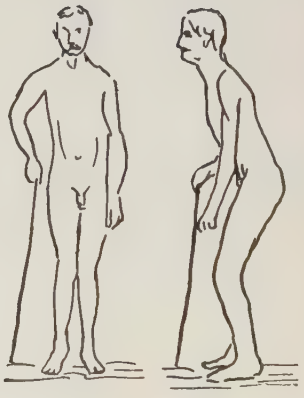


Fig. 204.

Fig. 200.—Congenital diplegia with athetosis in face and all extremities. (Church and Peterson.)

Fig. 201.—Posthemiplegic athethosis. (Barker, Monographic Medicine, 1916.)

Fig. 202.—Paraplegia in incomplete cord lesion from spinal fracture. Attitude of legs in bed: thighs adducted and crossed; bedsore. (Church and Peterson.)

Fig. 203.—Paraplegic gait. (Church and Peterson.)

Fig. 204.—Station in spastic paraplegia due to syphilitic myelitis. Rigidity; flexed knees; adducted thighs. (Church and Peterson.)

FIG. 205.—Spastic paraplegia: thighs and legs in flexion. Diagnosis of multiple sclerosis confirmed at autopsy. Patient is holding to a support above his bed by which he lifts himself. (Dejerine.)

FIG. 206.—Attitude of lower limbs in walking. Syphilitic paraplegia in a woman, aet. 29 years. (Dejerine.)

FIG. 205A.—Late contractures in case of spastic diplegia from double cortical lesions in infancy. Head and neck unaffected. Nutrition good. Limbs atrophied from lack of use; contractures from over action of stronger muscles. Reflexes not markedly increased. Dates from childhood. (Dejerine.)

FIG. 207.—Marked atrophy of lower limbs. Patient, aet. 39 years. Crush of conus terminalis and cauda equina by fracture. Patient commenced to gain power in lower limbs 18 months after injury. (Dejerine.)

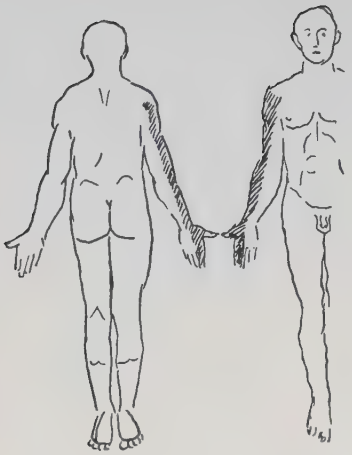


FIG. 208.—Syphilitic radiculitis of posterior roots of C. V.; VI and VII. Complete anæsthesia for all forms of sensation; no motor symptoms. (Dejerine.)

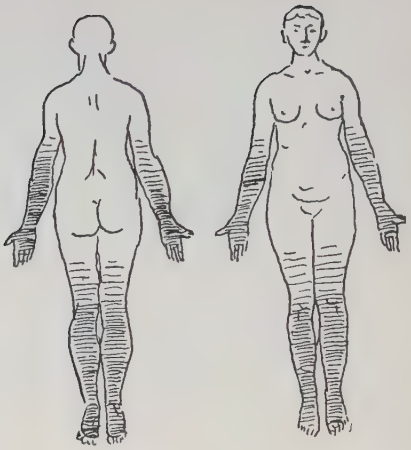


FIG. 209.—Post influenzal polyneuritis. Woman, aet. 28; marked loss of sense of position and bone sense; touch much diminished; pain, heat and cold showed delayed transmission with hyperæsthesia; astereognosis. Slight ataxia. Paralysis with atrophy of all four limbs. Cure in 14 months. Shading shows degree of anæsthesia. (Dejerine.)

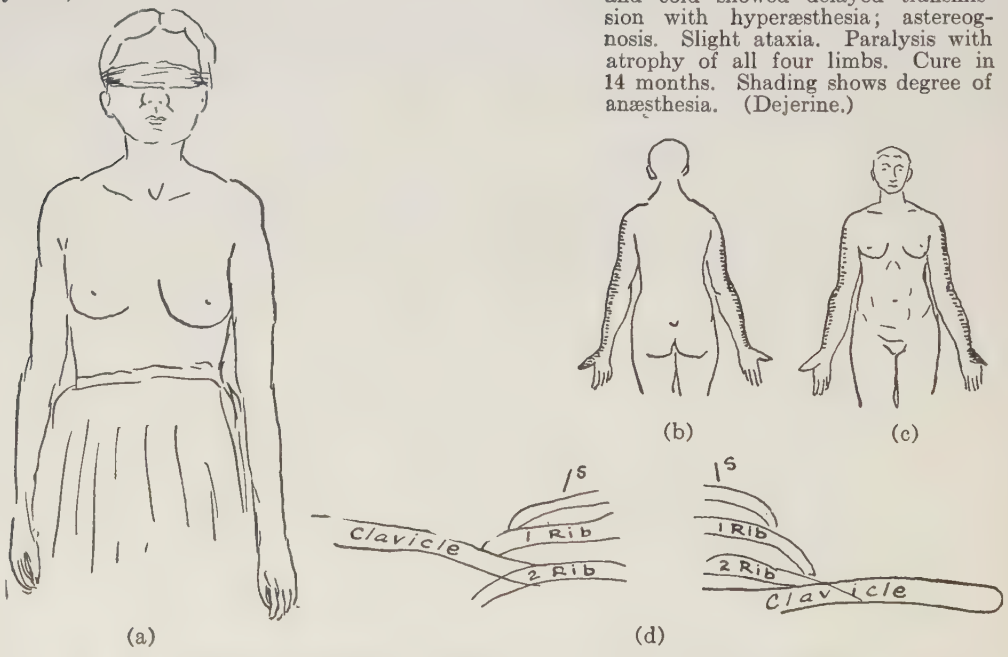


FIG. 210.—Lower motor neuron paralysis of C. V. and VI, due to cervical ribs (d). Paralysis with wasting of supra- and infra-spinatus, biceps, deltoid, brachialis, and brachioradialis. Woman of 35 years. Trouble dates from 1 year back. Pain and hypæsthesia in shaded areas. Operation on right side removing accessory rib; motor and sensory cure in one month. (a) Shows the attitude of paralyzed arms. Note the wasting of the deltoids and pronation of both forearms. (d) Radiograph of ribs; s, s, supernumerary ribs. (Dejerine.)

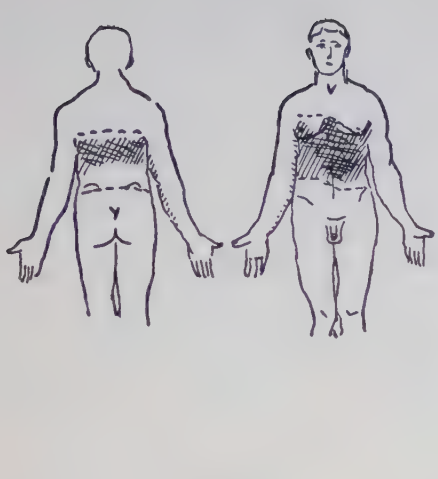


Fig. 211.

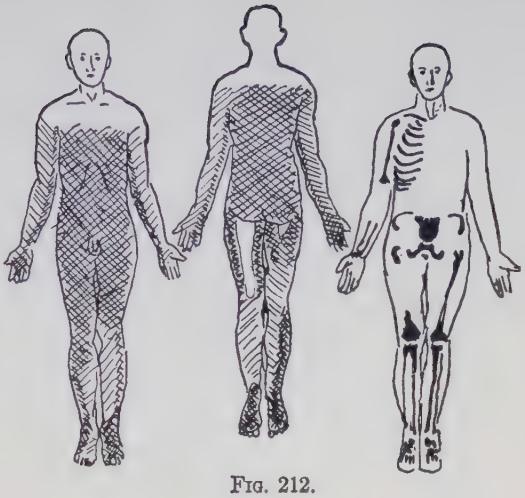


Fig. 212.

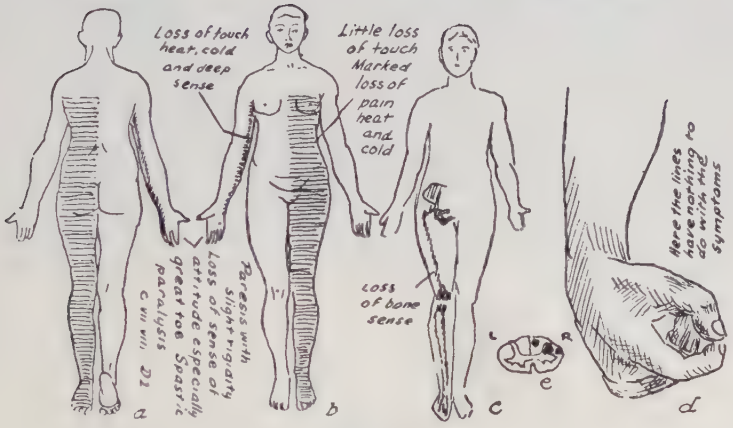

Loss of touch heat, cold and deep sense
Little loss of touch Marked loss of pain heat and cold
Paresis with slight rigidity
Loss of sense of attitude especially great toe. Spastic paralysis
C VII VIII D1
Loss of bone sense
Here the lines have nothing to do with the symptoms
a
b
c
d
e

Fig. 213.

Fig. 211.—Pre-ataxic tabes. Area of tactile anæsthesia. Man, aet. 33; syphilis at 21. Lightning pains for 3 months in lower limb. Slight paresis of bladder for same time. Patellar reflex normal on the right; weak on the left. Calcanean reflex lost on the right; weak on the left. No Argyll Robertson or Romberg sign. There was a corset sensation over rather a wider area than the anæsthetic area. (See broken line.) (Dejerine.)

Fig. 212.—Tabes dorsalis. Patient, aet. 47. History of syphilis. Loss of pain, thermic, tactile, and osseous sense in shaded areas, second sacral in left leg escapes. Degree of anæsthesia indicated by shading. Loss of patellar, calcanean, radial and olecranon reflexes. Argyll Robertson pupil, Romberg sign marked. Lightning pains in all 4 limbs; incontinence of urine; muscular hypotonia. Loss of sense of position in legs and right arm. Stereognostic sense lost in right hand, intact in left. (Dejerine.)

Fig. 213.—Hematomyelia (spontaneous) of 2 years' duration. (e), possible site of hemorrhage into seventh and eighth cervical and first thoracic segments. Spastic paralysis of *right* arm in muscles supplied by C. VII, C. VIII and D. I. (d); paresis with slight spasticity of right leg. Loss of touch, heat, and cold and deep sensibility on the inner border of the right arm. Loss of bone sense, and sense of position in the right leg, especially the great toe. On the *left* light touch is little affected; marked loss of pain, heat and cold. Brown Séquard type without the flaccidity. Anterior gray column escapes; lateral cerebrospinal tract only partly involved. (Dejerine.)

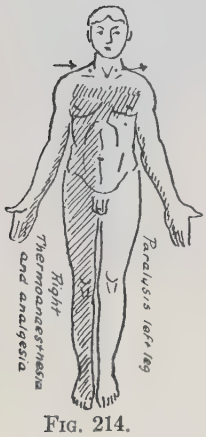


Fig. 214.

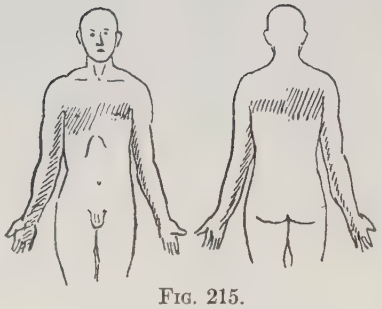


Fig. 215.

Fig. 216.

Fig. 214.—Incomplete transverse lesion of the cord, mainly affecting one lateral half. Brown Séquard syndrome. Ipsilateral paralysis, contralateral loss of pain, heat and cold. Shrapnel bullet, entry and exit shown by arrows. Six days after injury: Left lower limb completely paralyzed and flaccid; right lower limb could be moved at all joints. Abdominal and intercostal muscles paralyzed on both sides. Diaphragm normal. Left upper limb: Paralysis of intrinsic hand muscles and of all extensors and flexors of wrist, also of triceps. Right upper limb: Paralysis of intrinsic hand muscles and of flexors and extensors of fingers and thumb. Sensation: Loss of pain and temperature on the right lower limb and right half of the trunk to third rib, also down the medial side of the right arm in area of C. 7 and 8 and D. I: also across the left side of trunk in a zone, from the third rib to below the nipple and along the inner side of the left arm. Sense of position normal except in the two ulnar digits of the left hand. Bone sense lost in the lower limbs and lower trunk to 5th thoracic. Light touch lost in feet and ankles and lower third of leg. Six weeks later there was considerable movement of left leg; arms much better. Loss of pain, heat and cold unaltered.

Fig. 215.—Sensory loss in syringomyelia. Patient, aet. 55. Disease showed itself at 35, by atrophy of muscles of the right hand; at 50, there was atrophy of muscles of left hand, and later of the ulnar group of forearm muscles. Loss of sense of heat and cold, as in figure. Touch slightly affected. No loss of sense of position or of stereognostic sense. In area of 4th lumbar, on the left, there is loss of heat and cold without loss of touch.

Fig. 216.—Syringomyelia. Lower motor neuron paralysis. Patient, aet. 54, male. Disease showed itself at the age of 24 with loss of pain, heat and cold in both upper limbs. Compare Fig. 163. (Dejerine.)

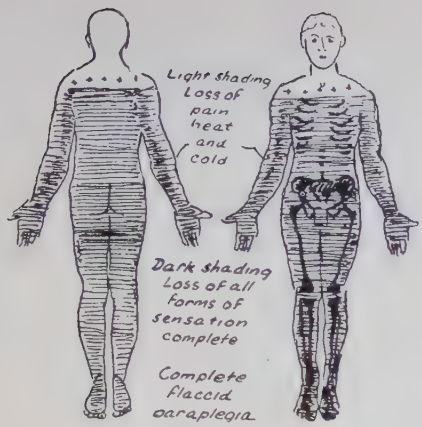


FIG. 217.

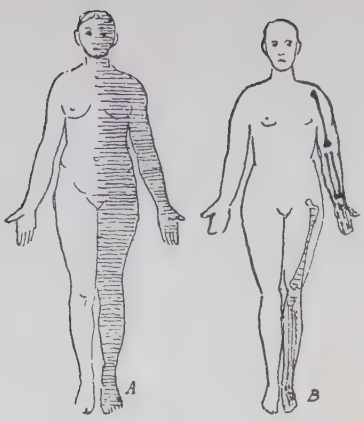


FIG. 218.

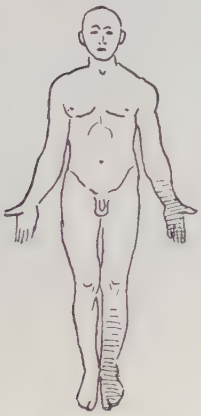


FIG. 219.

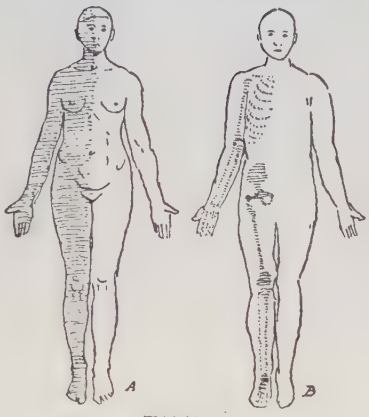


FIG. 220.

FIG. 217.—Fracture dislocation between 5th and 6th cervical vertebræ, crushing the 7th cervical segment of the cord (see Fig. 98). Complete anæsthesia and complete flaccid paralysis from 7th cervical segmental distribution down. Bone sense of sternum probably conveyed by clavicular branches of C. 3 and 4 nerves (C. 3 and 4). Light shading (C. 5 and 6) indicates loss of pain, heat and cold only (syringomyelic type). Note there is usually a strip of hyperæsthesia as indicated by x shading. It is not present in this case. (Case discussed in text, page 251.)

FIG. 218.—Sensory loss in capsular hemorrhage. Six months after the attack; patient aet. 51. Spastic hemiplegia on the left. Touch, pain and temperature sense much diminished, most affected toward extremities of the limbs, less affected near the middle line (see shading of figure). Sterognostic sense lost. Greatest loss of bone sense, in left upper limb as indicated by shading. Sense of passive position completely lost in upper limb; diminished below the knee. Hearing, taste, and smell intact. Right hemianopsia. At autopsy, thalamus uninjured; optic radiation, thalamocortical and pyramidal fibers involved. (Dejerine.)

FIG. 219.—Capsular type of hemianæsthesia. On the day after the attack there was slight left hemiplegia, marked hemianæsthesia for all forms of sensation, involving conjunctiva. Six weeks afterward, hemianæsthesia cleared up except at the extremities of the limbs as shown in figure. Bone sense affected. Complete astereognosis in hand and foot. Special senses intact. (Dejerine.)

FIG. 220.—Sensory loss in thalamic lesion; thalamic syndrome. Woman, aet. 52. *Motor symptoms:* Slight right hemiplegia. Hemiathetosis of hand and foot on right side. Right hemiataxia. Exaggerated tendon reflexes but *no Babinski*. *Sensory symptoms:* Touch, pain, and thermic sense diminished on right side. Localization and compass sense erroneous. Complete astereognosis. Taste, hearing and smell diminished. Sight normal. Sense of motion and passive position lost. Osseous sense as in Fig. B. (Dejerine.)

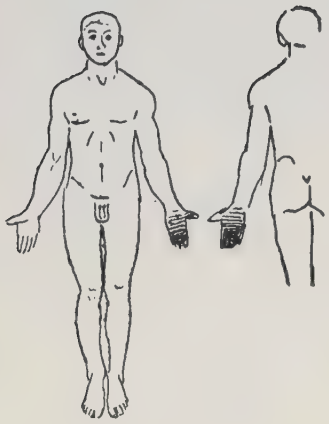


Fig. 221.

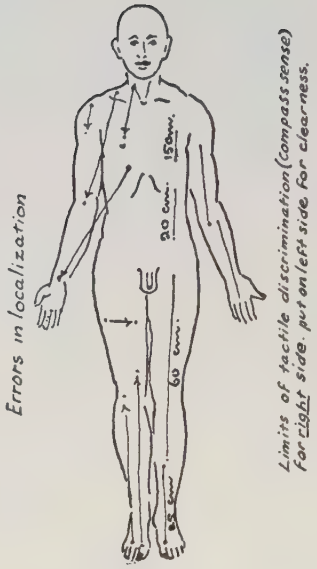

Errors in localization
Limits of tactile discrimination (compass sense)
For right side, put on left side for clearness.
150 mm.
90 mm.
60 mm.

Fig. 222.

Fig. 223.

Fig. 221.—Cortical anæsthesia. Tumor pressing on middle third of anterior and posterior central gyri. Partial left brachial monoplegia. Increased tendon reflexes of left arm and forearm. *Sensory loss:* Tactile sense most affected on dorsal surface of digits. Sense of localization much altered on back and front of fingers. Compass sense blunted. Sense of passive position of fingers completely lost. Astereognosis. Pain and thermic sense little affected. Bone sense not affected at all. Jacksonian epileptic attacks. (Dejerine.)

Fig. 222.—Left capsular lesion. Man, aet. 35. Enlargement of distance for compass test (shown for the sake of clearness on the left side of the figure but belonging to the right side on patient), and errors in localization. Arrows indicate error of localization. Light hemiplegia with hemianæsthesia on right side and right homonomous hemianopsia. Sense of position and stereognosis most affected. Superficial sense little affected. Choked disc. (Dejerine.)

Fig. 223.—Bulbopontine tumor. Woman, aet. 22. *Left side:* paralysis of VI, VII and VIII cranial nerves. Anæsthesia of left face for all forms of sensation. *Right side:* except on the face there is loss of pain, heat and cold; touch and muscle sense are little affected. (Dejerine.)

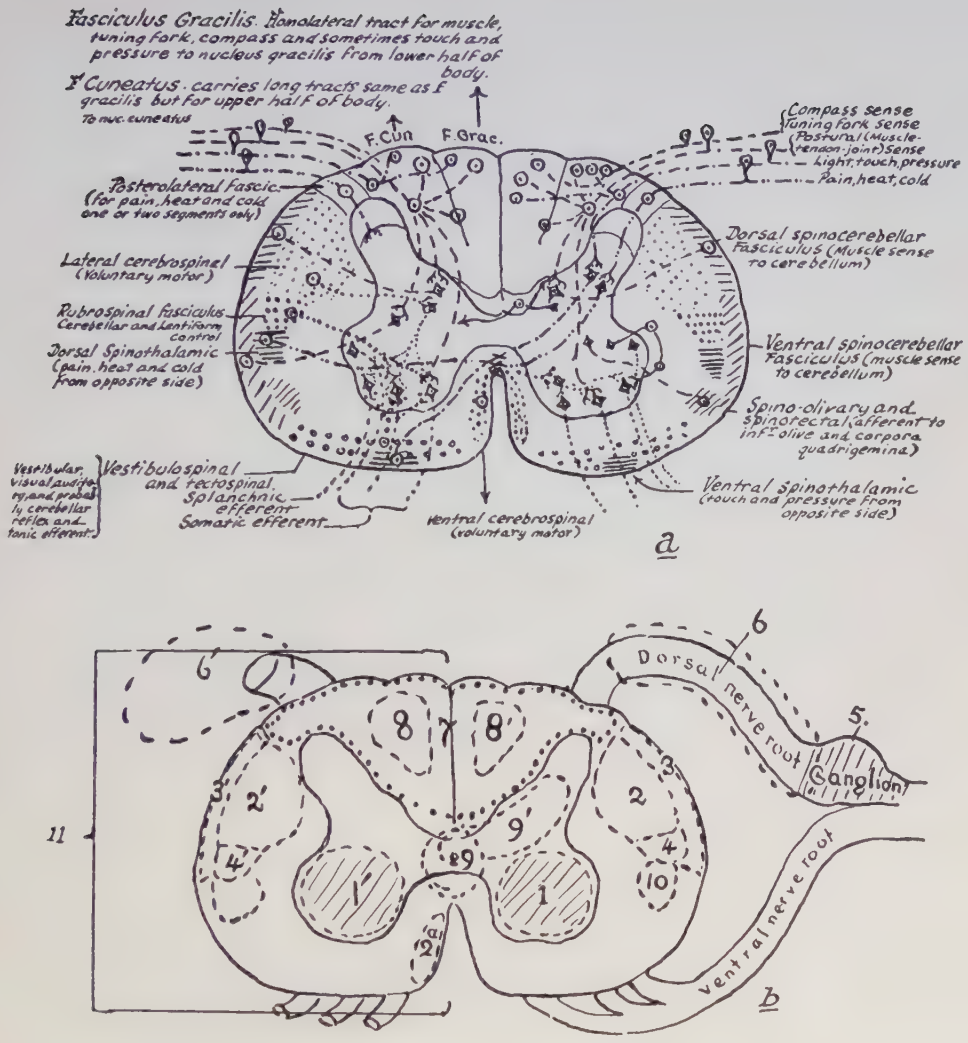

Fasciculus Gracilis. Homolateral tract for muscle, tuning fork, compass and sometimes touch and pressure to nucleus gracilis from lower half of body.
F. Cuneatus. carries long tracts same as F. gracilis but for upper half of body.
To nuc. cuneatus
F. Cun
F. Grac.
Compass sense
Tuning fork sense
(Postural (Muscle-tendon-joint) Sense
Light, touch, pressure
Pain, heat, cold
Posterolateral Fascic. (for pain, heat and cold one or two segments only)
Dorsal spinocerebellar Fasciculus (Muscle sense to cerebellum)
Lateral cerebrospinal (Voluntary motor)
Rubrospinal fasciculus Cerebellar and Lentiform control
Dorsal Spinothalamic (pain, heat and cold from opposite side)
Ventral spinocerebellar Fasciculus (muscle sense to cerebellum)
Spino-olivary and Spinotectal (afferent to inf. olive and corpora quadrigemina)
Vestibular, visual, auditory, and probably cerebellar reflex and tonic efferent
Vestibulospinal and rectospinal. Splanchnic efferent Somatic efferent
Ventral cerebrospinal (voluntary motor)
Ventral Spinothalamic (touch and pressure from opposite side)
a
Dorsal nerve root
Ganglion
Ventral nerve root
b

FIG. 224.—Diagram of more common cord lesions.

1, acute anterior poliomyelitis.
1, 1^1, progressive spinal muscular atrophy.
1, 1^1, 2, 2^1, amyotrophic lateral sclerosis.
2, 2a, tracts degenerating in hemiplegia (right).
2, 2^1, primary lateral sclerosis.
2, 3, 4, 2^1, 3^1, 4^1, syphilitic lateral sclerosis.
5, acute posterior poliomyelitis (herpes zoster).
6, Syphilitic radiculitis (Dejerine).
6, 6^1, primary lesion in tabes dorsalis.
7, secondary lesion in tabes dorsalis.
8, 8^1, primary posterior sclerosis.
9, 9^1, early lesions in syringomyelia.
10, lesion (such as localized tubercle) causing loss of pain, heat and cold on opposite side of body below lesion.
2, 3, 4, 7, 2^1, 3^1, 4^1, combined sclerosis. Friedreich's ataxia.
11, half lesion, Brown Séquard paralysis.
Complete transverse lesion, myelitis, myelomalacia, trauma, tumor.

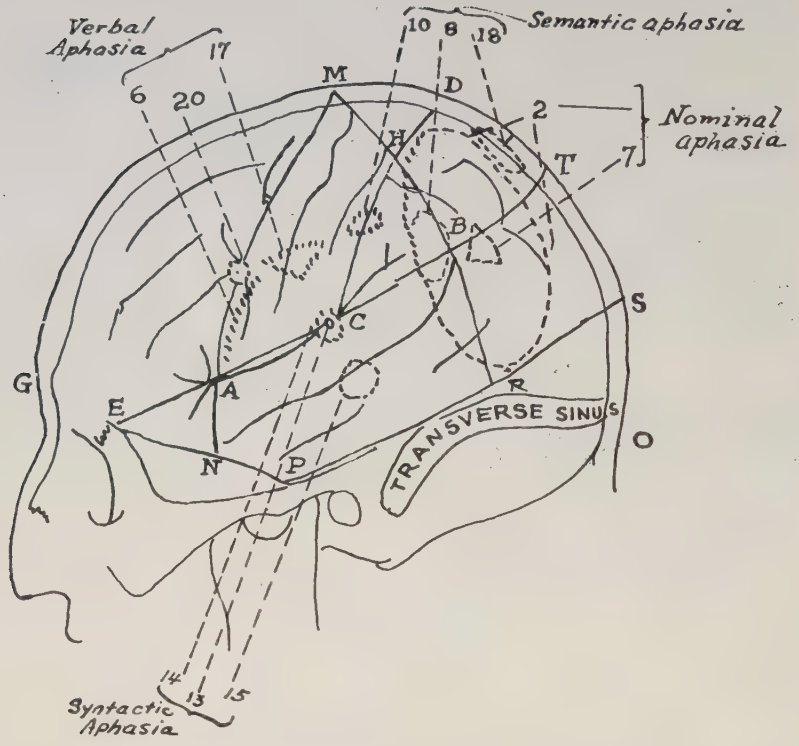
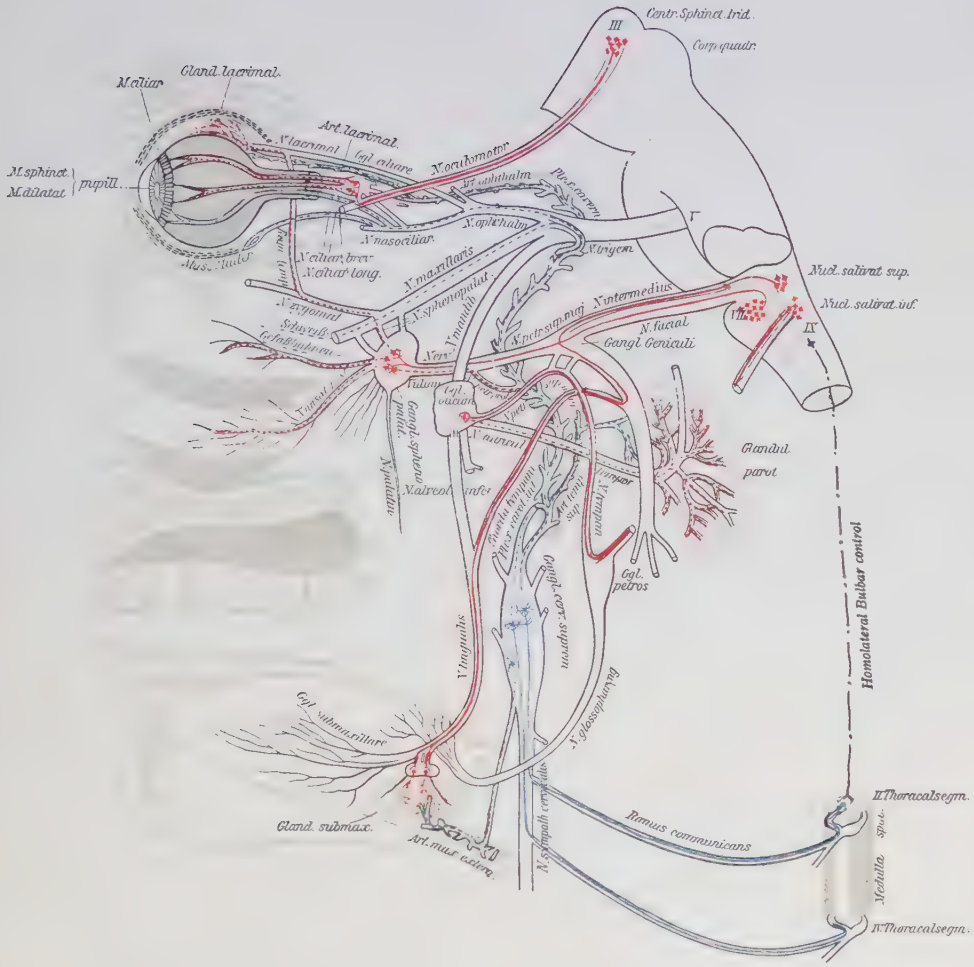


FIG. 225.—Approximate sites of lesions described and classified in Head's *Aphasia and Kindred Disorders of Speech* (1926, The Macmillan Co.), compiled from Head's pictures and adapted to the figure of cranio-cerebral topography in Cunningham's Textbook of Anatomy. The numbers refer to the numerical order of Head's cases in his work on Aphasia. Chiene's lines in cranio-cerebral topography:

G, glabella.
O, external occipital protuberance (inion).
M, midpoint between G and O.
T, midpoint between M and O.
S, midpoint between T and O.
P, root of Zygoma.
E, external angular process (Zygomatic process).
N, midpoint between E and P.
R, midpoint between P and S.
NM is the shortest line on the scalp between N and M. Similarly the lines RM, ET, PS are drawn as indicated.
A and B are the points where the lines NM and RM cut ET.
C is the midpoint between A and B.
CD is drawn parallel to AM.

The central sulcus runs parallel with AM and midway between AM and CD. AM is over the precentral sulcus; CD over the post central sulcus; B marks the angular gyrus. The triangle CHB marks the supramarginal gyrus.

For full description of Chiene's method of cranio-cerebral topography see Cunningham's Textbook.



Schema of the parasympathetic and sympathetic systems in the head and neck.
From Edinger after F. W. Müller (slightly modified, w. k.).

Parasympathetic—red.
Sympathetic—blue.
Preganglionic fibers—continuous lines.
Postganglionic fibers—broken lines.

The long diagram opposite is recommended to the close attention of all earnest students of the nervous system.

It is designed as a means of visualizing and reviewing the anatomy and chief functions of the best known nerve tracts.

It may be separated from the book and mounted on a roller at each end, so that in reading downward or upward, as the tract may require, it may be unrolled at one end and rolled up at the other.

Should the student desire to make it clearer by coloring the lines, red is recommended for efferent (descending) tracts, blue for tracts for pain and temperature, purple for tracts for muscle sense, and green for cerebellar afferent tracts. Inks, not chalks, should be used.

INDEX

Abdominal viscera, center for, 247
Abducent nerve, 39, 89, 342
Aberrant pyramidal fibers, of Déjerine, 43, 45, 46, 50, 64, 91, 93, 94, 127
Abnormal attitudes in cerebellar lesions, 269, 317
Abscess of cerebellum, 319
Accessory fillet, 64
Accessory nerve, 34, 78, 120, 122
Accessory olive, 32, 83
Absence of cerebellar hemisphere, 370
Activity, cortical, relation of to body sense, 217
Adiadochokinesis, 268, 397
Adduction reflex, 232
Afferent neurons, 8, 13
Afferent paths, auditory, 21
  cerebellar, 21, 22, 315, 391
    affections of, 320
  for head, 17
  spinocerebellar, 21
  vestibular, 391
  visual, 21
Afferent tracts, 16
  to cerebellum, 21
  to inferior olive, 167
Agenesis of cerebellum, 319
Agnosia, 283
Alcoholic peripheral neuritis, 406
Alexia, 162
Amyotrophic lateral sclerosis, 245, 302, 421
Anæsthesia, 180
  cortical, 430
  following section of posterior nerve root, 145
  in tabes dorsalis, 201
Analgesia, 180
Ankle clonus, 185
Ansa lenticularis, 55
Antero-internal bulbar syndrome, 411, 412
Anterolateral columns, 58, 60
  degeneration in, 72
Anterolateral fasciculus proprius, 75
Aphasia, 182, 235, 279, 286, 432
  Head's classification of, 287
  nominal, 288
  semantic, 288
  syntactic, 288
Aphasia, verbal, 287, 288
Apraxia, 182, 286
  clinical cases of, 284
  cortical, 284
  ideational, 284
  motor, 283, 284, 409, 410
  subcortical, 284
Aqueductus cerebri, 211
Arcuate fibers, deep, 79
  external, 34
  internal, 16, 31, 81
  superficial, 79, 80, 83
Arcuate nucleus, 34
Areas, association, 174
  calcarine, focal symptoms in, 325
  cutaneous, segmental arrangement of, 383
  localizing, 375, 402, 403
  motor, 49, 375, 376, 402
  olfactory, 162
  prefrontal, 279
  pyriform, 325
  radicular, 198
  segmental, 198
  sensory, 183, 190, 217, 218, 219, 220, 314, 375, 376, 379, 402
  silent, 174, 326
  skin, sensory, 183
Arteries, cerebellar, lesion of, 413
  cerebral, 403
    degeneration, 366
    embolism of, 234
    thrombosis of, 234
  cortical, 403
  involved in paralysis, 237
  lenticulostriate, 237
  spinal, thrombosis of, 411
  vertebral, thrombosis of, 412
Ascending degenerations, 71
Association areas, 174
Association fibers, 174, 403
  lesions of, 323
Association neurons, 13, 67
Asthenia, in cerebellar lesions, 265
Asynergia, Babinski's, 266
  cerebellar, 266, 394
Ataxia, 181, 220
  cerebellar, 181, 182, 265, 317
    analysis of, 266

Ataxia, cerebellar, asynergia in, 266
decomposition movement in, 266
deviation in, 268
dysmetria in, 267
familial, 319
hereditary, 319
tremor in, 267
cerebral, 283
Friedreich's, 205, 245, 319, 320, 422
following section of posterior nerve roots, 145
in tabes dorsalis, 202
spinocerebellar, 320
vestibular, 181
Athetoid movements, 216, 232
Athetosis, 131, 273
posthemiplegic, 425
Atonia, cerebellar, 265, 393
Atrophy, cerebellar, 319, 396, 397
muscular, following section of posterior nerve root, 146
of globus pallidus, 400
of inferior olive, 370
progressive muscular, 405, 406
Attitude, in cerebellar lesions, 264, 269, 317
Auditory nerve, 23, 35, 109
Auditory nuclei, path from to eye muscles, 157
Auditory paths, 21
Auditory radiation, 157, 158
Auditory sense, paths for, 18
Aura, sensory, 234
Autonomic centers in cord, 246
Autonomic control in medulla and pons, 210
Avulsion of motor nerve roots, 65
Axons, 6
degeneration of, 9, 11
Purkinje, 167, 168
pyramidal, disease near, 188

Babinski's asynergia in cerebellar ataxia, 266
phenomenon, 131, 184, 232
surréflectivité hyperalgésique, 258
Bandalette, of Hoche, 70, 138
Barany's pointing test, 269
Basal ganglia, 271, 372
Basilar portion of pons, 36, 85
lesions of, 297, 298, 320
Basis mesencephali, 37
Basis pedunculi, 37, 45, 212
lesions of, 297
Bechterew, nucleus of, 163
Benedict's syndrome, 418
Betz cells, 14, 19, 50, 102, 127
Bilateral paralysis, upper motor neuron, 300
Birth palsy, 406
Bladder control, center for, 247
Blindness, 160
cortical, 283
mind, 283
word, 162
Body, geniculate, 47
Nissl, 345
of nerve cell, 6
restiform, 31, 35, 82, 83, 210, 338
lesions of, 396
section through, 74
trapezoid, 37, 38
Body sense, 14
relation of cortical activity to, 217
Bone sense, test for, 15
Brachium conjunctivum, 22, 42, 43, 44, 45, 46, 52, 85, 87, 91, 93, 94, 95, 96
decussation of, 45, 46, 93
dissection of, 340
lesions of, 297, 320
Brachium pontis, 37, 86, 167
lesions of, 320
Brain, convolutions of 373, 374
efferent paths from, 20
fibræ propriæ of, 173
internal anatomy of, 3
motor tracts in, 19
sensory tracts in, 16
softening of, 127
Brain stem, fœtal, 74
reflexes in, 173, 377
sections of, 56
sensory tracts in, lesions of, 308
ventral surface of, to show origin of cranial nerves, 336
Brown-Sequard paralysis, 107, 152, 255, 258, 322, 388, 389
Brown-Sequard syndrome, 230, 428
Bruce, septomarginal bundle of, 70, 138
Bulb, *see* Medulla oblongata
Bulbs, end, 347
Bulbar paralysis, 230, 245
Bulbar syndrome, antero-internal, 411, 412
retro-olivary, 412, 413, 414
Bulbocavernous reflex, 184
Bulbopontine form of poliomyelencephalitis, 404
tumor, 431
Bundle, cornucommisural, 70
mammillothalamic, 52
medial longitudinal, 84
Monakow's, 95
olivocerebellar, 167
septomarginal, 66, 70, 138
Burdach, column of, 31

Calcarine area, focal symptoms in, 325
lesions of, 161

Canal, central, 27, 29, 59, 60, 62
Cancer of spinal column, 64
  pelvic, 152
Cap of gelatinous substance, 59
Capsular hemianæsthesia, 429
  hemiplegia, 429
  hemorrhage, 194, 429
  lesions, 221, 230, 231, 296, 313, 431
  paralysis, 237
  softening, 237, 238
Capsule, internal, 19, 50, 63, 127, 372
Caput columnæ posterioris, 26
Carcinoma, compression by, 367
  of spinal column, 64
  pelvic, 152
Cardiac center, 247
Catalepsy, cerebellar, 267
Cauda equina, lesions of, 225, 253, 425
Caudate nucleus, 55, 272
Cells, anterior, fœtal, 75
  Betz, 14, 19, 50, 102, 127
  Golgi, type I., 7, 8, 9
    type II., 9, 26, 55, 345
  intercalated, 70
  motor neuron, 60, 62
  nerve, 5, 6
  Purkinje, 8, 37, 167, 168, 345
  pyramidal, 188, 344, 345
Centers, 11
  autonomic, in cord, 246
  cardiac, 247
  ciliospinal, 246
  for abdominal viscera, 247
  for bladder control, 247
  for form, 54
  for posture, 54
  for smell, 282
  for sweating, 246
  for tear secretion, 246
  for touch, 54
  genito-urinary, 247
  in medulla oblongata, 33
  pupillodilator, 246
  rectoanal, 247
  reflex, 246, 248, 377
Central canal, 27, 29, 59, 60, 62
  convolutions, focal symptoms in, 323, 324
  tegmental tract, 83, 95, 97, 167
Centripetal neurons, 8
  fibers of optic nerve, 111
Cerebellar afferent paths, 21, 22, 391
  artery, lesion of, 413
  asynergia, 266, 394
  ataxia, 181, 182, 265, 266, 267, 268, 317
    hereditary, 319
  atonia, 265, 393
  atrophy, 396, 397
Cerebellar catalepsy, 267
  convulsions, 318
  dysmetria, testing for, 394, 395, 396
  dys-synergia, 394
  efferent paths, 22, 42, 167, 168, 391
    affections of, 320
  folium, section through, 341
  gait, 269, 270, 318, 396
  hemisphere, 166
    absence of, 370
  hemorrhage, 320
  hypotonia, 265, 393
  lesions, 97, 170, 317, 319, 320, 392, 393, 394, 395
    attitudes in, 264, 269, 317
    eye symptoms in, 318
    knee jerk in, 318
    nystagmus in, 318
    reflexes in, 270
    skew deviation in, 318
    speech in, 270, 318
    standing in, 269
    symptoms in, 232, 263, 265, 268, 271
  mechanism, 21
  neurons, 14, 166
  nuclei, 42
  paresis, 265
  paths, affections of, 320
  softenings, 320
  tract, descending, 96
  tremor, 397
Cerebello-pontine angle, lesions in, 321
Cerebello-thalamo-striatepath, 171
Cerebellum, 163, 261, 315
  abscess of, 319
  absence of, 169
  afferents to, 21, 315
  agenesis of, 319
  anatomy of, 165
  atrophy of, 319
  cirrhosis of, 319
  connections of, 163
  diseases of, 319, and *see* Cerebellar lesions
  functions of, 163, 169, 261, 316
  hemispheres, 315, 316
  hypoplasia of, 319
  lesions of, *see* Cerebellar lesions
  physiology of, 165, 169
  traumatisms of, 320
  tumor of, 319
Cerebral arteries, 403
  degeneration, 366
  embolism of, 63, 234
  thrombosis of, 234
Cerebral association neurons, 13
Cerebral ataxia, 283
Cerebral cortex, arteries of, 403

Cerebral cortex, convolutions of, 402
degeneration in, 366
efferent paths from, 19
fissures of, 402
lesions of, 274
localizing areas of, 375, 402, 403
motor areas in, 49, 375, 376, 402
sensory areas of, 219, 375, 376, 402
silent areas of, 174, 326
softening of, 366
structure of, 343
Cerebral hemisphere, 282, and *see* Cerebral cortex
association areas of, 174
association fibers of, 174
commissural fibers of, 174
Cerebral localization, 127
Cerebral peduncles, section through, 96, 360
Cerebral monoplegia, 236, 424
Cerebrospinal fasciculus, 20, 30, 62
Cerebrospinal nervous system, 362
Cerebrospinal sclerosis, 302
Cerebrospinal tract, 20, 30, 64, 76, 77, 78, 129
Cervical cord, gliosis of, 310
section through, 347
Cervical enlargement, 60, 74, 76, 120
Cervical nerves, 78, 88, 333
Cervical ribs, 426
Cervical segments, 28, 61, 64, 67, 70, 73, 77, 120, 249, 250, 251, 333, 335, 349
Cervical vertebræ, fracture dislocation of 429
Cervix columnæ posterioris, 26
Charcot's joint disease, 390
Chipault's rule for relation of cord segment to spinous processes, 259
Chorda tympani nerve, 109
gustatory fibers of, 156
Chorea, Huntington's, 273, 322
of children, 274
of pregnancy, 274
Sydenham's, 274
Choreiform movements, 216, 232
Chorioid plexus, 35
Chromatolysis, 11, 146, 345
Ciliospinal center, 246
Cirrhosis of cerebellum, 319
Clarke's column, 27, 60
Clava, 31
Clonic convulsions, 187
Clonus, ankle, 185
Cochlear nerve, 38, 85, 109, 156, 166, 384
Cold, fibers for, 137
neurons for, 103, 104, 151
sensation of, 135, 304
test for, 14, 15, 142
tracts for, 17
Collateral, 363
Colliculus facialis, 89
Colliculus inferior, 44, 91, 93, 341, 342, 357, 358
superior, 46, 48, 94, 96, 159, 341, 359, 360
Columns, anterior, 59, 60, 61
anterolateral, 58, 60, 72
Burdach's, 31
Clarke's, 27, 60
degeneration in, 72
Goll's, 31
gray, 58, 59, 60, 61
level of lesions in, 259
Lissauer's, 26, 58
posterior, 57, 59
sclerosis of, 385
posterolateral, 58, 61, 75
white, 57, 58, 60
Coma and early flaccidity, 238
Combined sclerosis, 205, 244, 302, 408
Comma tract, 66, 138
Commissural fibers, 174
lesions of, 323
Commissure, gray, 24, 27
Gudden's, 158
white, 58
Common sensation, paths of, 17, 19
Compass sense, tract for, 16
Conducting paths, sensory, lesions in, 306
Congenital diplegia, 236, 424
Congenital double hemiplegia, 423
Conjugate deviation of eyes, 89
Contractures, 194, 425
Conus terminalis, lesions of, 425
Convolutions, cerebral, 373, 374, 402
focal symptoms in, 323, 324
Convulsions, cerebellar, 318
clonic, 187
generalized cortical, 187
lower motor neuron, 187
tonic, 187
Coördinating mechanism, 21
Cord, *see* Spinal cord
Cornucommissural bundle, 70
tract, 68, 138
of Dufour, 139
Corpora, *see under* Corpus
Corpus callosum, 175, 403
focal symptoms in, 326
dentatum, lesions of, 320
mammillare, 52
quadrigeminum, 171
lesions of, 68, 171
striatum, 24, 54, 171, 271
lesions of, 321
physiology of, 172
trapezoideum, 37, 38, 39, 85, 86, 91, 110
Cortex, afferent paths from to cerebellum, 22
degeneration in, 366

Cortex, efferent paths from, 19
lesions of, 190, 217, 220, 274, 314, 323
localizing areas in, 218, 375, 402
motor areas of, 49, 375, 376
sensory areas of, 190, 217, 218, 219, 220, 314, 375, 376
silent areas of, 174, 326
softening of, 366
structure of, 343
Cortical anæsthesia, 431
apraxia, 284
arteries, 403
blindness, 283
convulsions, 187
deafness, 283
lesions, 230, 233, 234, 296
monoplegia, 236
paralysis, 235, 283
softening, 236
vessels, blocking of, lesions due to, 234
Corticocerebellar fibers, 166
Corticopontine system, 34
Corticoponto-cerebellar neurons, 166
Corticothalamic fibers, 53
Cranial nerves, *see under* Nerves
Cremasteric reflex, 184
Crises, tabetic, 189
Crossed paralysis, 180, 298
Crossed pyramidal tract, 20, 30
Cutaneous areas, segmental arrangement of, 383
Cutaneous nerves, lesions of, 196, 307
myelinated, 14
unmyelinated, 15
Cutaneous reflexes, 184, 185, 194

Deafness, 158
cortical, 283
mind, 283
Decerebrate rigidity, 190, 290
Decomposition movement in cerebellar ataxia, 266
Decussation, Forel's, 46, 49, 94
fountain, 49
Meynert's, 49, 94, 95
of brachia conjunctiva, 45, 46, 93
of pyramids, 30, 62, 64, 78, 99, 103, 230, 350, 351
rubrospinal, 46, 49, 94, 95, 359
tectospinal, 49, 359
Deep sensibility, 15, 135
test for, 15, 143
Defense reflex, 193
Degenerated nerve tract, 346
Degenerations, ascending, 71
descending, 69
in posterior column, 70
Degenerations, following cerebral artery lesion, 366
following section of nerve roots, 145
in cerebral cortex, 366
in spinal cord lesions, 69, 70, 72, 368, 369
in tabes dorsalis, 203
lenticular, 272, 321, 397, 398
of axon, 9, 11
of cell bodies, 9
of globus pallidus, 399
of pallidal system, 401
of pyramidal tract, 133
of rubrospinal tract, 370
of spinal cord, later, 72
in tumor of dura, 369
phenomena of, 9
striatal, 401
Wallerian, 10, 146, 346
Degeneration methods, 62, 63, 66
Deiters' nucleus, 23, 36, 42, 68, 69, 84, 164, 209, 352, 364
Déjerine, aberrant pyramidal fibers of, 43, 45, 50, 64, 91, 93, 94, 127
thalamic syndrome of, 313
Dendrites, 6
Dentate nucleus, 84
Dermatoses corresponding to dorsal nerve roots, 378
Descending association neurons, 67
cerebellar tract, 96
degenerations, 69, 70
Destructive lesions of neurons, 187
Deviation, conjugate, of eyes, 89
in cerebellar ataxia, 268
skew, of eyes, 263
spontaneous, of arm, 268
Diaschisis, 182, 194, 195, 263
Diencephalon, section through, 49
Diplegia, 180
congenital, 236, 425
in children, 241
spastic, 241, 426
Direct pyramidal tract, 20, 30
Discrimination, tactile, 14, 304
Disseminated sclerosis, 321
Dorsal nerve roots, 363
dermatoses corresponding to, 378
skin areas corresponding to, 378
Dorsal nucleus of spinal cord, 27
Dorsal spinocerebellar tract, 31, 60, 76, 165
Dorsolateral thalamic nucleus, 41
Dorsoventral pontile syndrome, 415, 416
Double hemiplegia, 241, 423
Double porencephaly, 237
Drop wrist, 404
Dufour, cornucommissural tract of, 139
Dura, tumor of, 72

Dura, degeneration in spinal cord following, 360
Dysæsthesia, 180
Dysarthria, 286
Dysdiadochokinesis, 268
Dysmetria, cerebellar, 267, 394, 395, 396
Dys-synergia, 394

Efferent neurons, 12
  lower, 125
  somatic, 117, 125
  splanchnic, 117, 125
Efferent nucleus, splanchnic, 100, 123
Efferent paths, cerebellar, 22, 42, 167, 168, 316, 391
  affections of, 320
Efferent paths, cerebral, 19, 20
  spinal, 20
  vestibular, 164
Efferent tracts, 16
  from vermis, 167
Embolism, cerebral, 63, 234
Eminentia medialis, 89
Encephalitis lethargica, 94, 274
End bulbs, 347
End organs, receptive, 8, 9
Endogenous fibers, 365
  neurons, 66, 67
Ependyma, 35
Epicritic sense, 14, 135
  test for, 142
Epigastric reflex, 184
Epilepsy, Jacksonian, 233
Equilibration, mechanism of, 164
  organs of, 23, 84
Equilibratory mechanism, 23
Erb's syphilitic paraplegia, 244
Essential thalamic organ, 19, 53, 140
  relay to, 213
Eupraxia, 285
Extensor response, plantar, 184
Extrapyramidal motor mechanism, 171, 261
Eye symptoms in cerebellar lesions, 318
Eye-turning muscles, reflex path from auditory nuclei, 157
Eyes, conjugate deviation of, 89
  skew deviation of, 263

Face, muscle sense complex for, 106
  vegetative innervation to, 433
Facial fibers for cold, heat, and pain, 137
Facial monoplegia, 236
Facial nerve, 90, 108, 123, 342
  ganglia of, 198
  lesions of, 311
  nucleus of, 39, 90, 100, 103, 123
Facio-brachial monoplegia, 423
Familial periodic paralysis, 303
  spinocerebellar ataxia, 319
Fasciculus, cuneatus, 24, 60, 61, 73
  gracilis, 24, 60, 61, 71, 76
  interfascicularis, 66, 70, 138
  medial longitudinal, 33, 39, 44, 46, 47, 80, 82, 87
  posterolateral, 61
  proprius, 13, 75
  septomarginal, 76, 77
  solitarius, 30, 35, 90, 156
  spinocerebellaris, 79, 91
  spino-olivary, 68, 167
Fibers, association, 174, 403
  commissural, 174
  deep, 75
  for cold, 137
  for heat, 137
  for pain, 137
  frontopontine, 37, 51, 96
  myelination of, 37
  olivocerebellar, 83
  posterior, 75
  pyramidal, 37, 50, 85
  superficial, 75
  temporopontine, 37, 51, 96
  unmyelinated, 7
  upper motor neuron, 50
Fibræ propriæ, 173
Fibrillar twitchings, 187
Field, oval, 76, 128
  visual, 387
Figure **A**, 119, 129, 136, 138, 139, 161, 168, 284
B, 119, 136, 138, 139, 164, 168, 175, 226, 285
C, 120, 129, 136, 138, 139, 164, 165, 168, 285
D, 120, 122, 139, 164, 165, 285
E, 122, 128, 139, 164, 165, 207, 208, 209, 286
F, 120, 122, 123, 136, 150, 155, 164, 165, 207, 208, 209, 326
G, 128, 136, 150, 163, 164, 165, 167, 168, 207, 208, 209, 326
H, 120, 123, 124, 128, 136, 150, 157, 164, 168, 207, 208, 326
I, 124, 136, 140, 150, 157, 165, 166, 168, 207, 326, 327
J, 121, 128, 136, 139, 150, 157, 158, 168, 327
K, 121, 150, 157, 168, 327
L, 121, 127, 157, 159, 166, 168, 209, 212, 327
M, 136, 139, 141, 157, 158, 168, 211, 213, 327
N, 328
O, 328

Figure P, 328
Q, 328
R, 328
Figure 1, 24, 26, 27, 28, 61, 62, 120, 129, 333
2, 24, 25, 26, 27, 60, 61, 118, 120, 333
3, 24, 25, 26, 27, 59, 119, 334
4, 24, 25, 26, 27, 56, 57, 58, 118, 119, 334
5, 24, 25, 26, 27, 56, 335
6, 11, 12, 20, 24, 25, 60, 61, 120, 335
7, 11, 12, 20, 21, 24, 25, 27, 59, 60, 119, 325, 335
8, 11, 12, 20, 24, 25, 59, 119, 336
9, 11, 12, 20, 24, 25, 119, 324, 336
10, 12, 19, 20, 30, 32, 34, 40, 120, 121, 122, 123, 124, 156, 336
11, 12, 21, 28, 29, 31, 34, 38, 39, 80, 89, 120, 123, 124, 156, 157, 163, 325, 337
12, 12, 20, 29, 34, 39, 40, 47, 89, 90, 91, 92, 99, 120, 121, 122, 123, 124, 156, 324, 337
13, 12, 20, 29, 39, 40, 47, 99, 120, 122, 123, 124, 156, 325, 337
14, 12, 20, 29, 32, 34, 35, 40, 41, 81, 83, 120, 122, 123, 155, 338
15, 21, 29, 31, 32, 33, 34, 35, 41, 80, 81, 82, 88, 155, 338
16, 29, 36, 37, 40, 124, 128, 339
17, 36, 37, 38, 43, 91, 128, 339
18, 44, 91, 92, 121, 340
19, 42, 308, 340
20, 42, 167, 341
21, 37, 42, 167, 341
22, 44, 45, 50, 86, 92, 93, 121, 341
23, 46, 48, 86, 92, 98, 121, 341
24, 39, 40, 89, 90, 91, 123, 342
25, 342
26, 49, 52, 53, 96, 97, 342
27, 7, 12, 14, 19, 127, 174, 274, 275, 343
28, 343
29, 6, 7, 20, 344
30, 6, 7, 12, 344
31, 6, 7, 8, 344
32, 6, 7, 8, 22, 345
33, 6, 7, 9, 345
34, 345
35, 10, 345
36, 10, 11, 345
37, 10, 346
38, 10, 346
39, 10, 346
40, 10, 346
41, 10, 346
42, 10, 346
43, 8, 12, 13, 136, 138, 346
Figure 44, 8, 9, 346
45, 8, 9, 136, 347
46, 8, 9, 136, 347
47, 8, 9, 347
48, 8, 9, 138, 347
49, 8, 9, 136, 347
50, 74, 165, 347
51, 74, 75, 139, 165, 347
52, 74, 75, 139, 165, 348
53, 9, 11, 20, 24, 27, 28, 32, 74, 75, 78, 118, 129, 348
54, 11, 20, 21, 24, 27, 28, 32, 74, 76, 77, 78, 119, 129, 165, 348
55, 11, 20, 24, 28, 30, 32, 74, 76, 78, 120, 129, 349
56, 20, 26, 28, 30, 32, 35, 41, 62, 74, 77, 120, 122, 129, 349
57, 20, 26, 29, 31, 32, 34, 35, 41, 62, 74, 77, 78, 122, 129, 350
58, 20, 26, 29, 30, 31, 32, 34, 41, 74, 77, 78, 122, 123, 128, 351
59, 12, 19, 20, 21, 26, 29, 30, 31, 32, 33, 34, 35, 36, 41, 74, 79, 80, 90, 120, 122, 123, 139, 155, 351
60, 12, 19, 20, 21, 22, 23, 26, 29, 30, 31, 32, 33, 34, 35, 38, 41, 42, 74, 80, 82, 83, 84, 90, 91, 109, 123, 128, 163, 167, 352
61, 20, 33, 38, 41, 74, 85, 86, 90, 91, 109, 156, 157, 163, 353
62, 21, 22, 33, 36, 38, 39, 40, 41, 43, 74, 85, 86, 87, 90, 91, 120, 124, 128, 354
63, 19, 22, 33, 36, 37, 38, 39, 40, 41, 42, 43, 74, 85, 86, 87, 88, 89, 90, 91, 120, 124, 140, 157, 355
64, 19, 32, 33, 36, 37, 38, 41, 43, 44, 50, 72, 74, 86, 91, 92, 121, 127, 356
65, 33, 36, 37, 38, 41, 44, 45, 74, 91, 93, 128, 357
66, 22, 33, 37, 38, 41, 44, 45, 46, 74, 91, 92, 93, 121, 358
67, 22, 33, 37, 38, 46, 47, 49, 74, 94, 95, 96, 97, 98, 112, 168, 359
68, 20, 22, 33, 38, 46, 47, 48, 49, 52, 74, 82, 86, 89, 91, 93, 96, 97, 112, 121, 159, 360
69, 12, 13, 14, 15, 16, 17, 20, 21, 22, 23, 27, 28, 32, 33, 36, 47, 57, 58, 60, 61, 66, 70, 71, 103, 105, 107, 112, 118, 135, 136, 138, 140, 141, 149, 150, 152, 164, 165, 173, 207, 225, 361
70, 12, 13, 14, 16, 17, 18, 19, 20, 21, 22, 23, 118, 173, 362

Figure 71, 6, 9, 13, 186, 363
72, 6, 9, 13, 363
73, 9, 13, 363
74, 6, 9, 13, 57, 67, 138, 173, 186, 363
75, 13, 57, 118, 173, 186, 363
76, 13, 67, 118, 119, 173, 186, 363
77, 10, 11, 68, 69, 70, 84, 364
78, 10, 32, 64, 65, 67, 69, 70, 83, 139, 167, 223, 365
79, 10, 19, 20, 30, 31, 45, 46, 50, 51, 63, 64, 70, 102, 103, 127, 128, 129, 133, 134, 236, 239, 366
80, 10, 64, 65, 66, 105, 138, 139, 146, 367
81, 10, 21, 62, 69, 70, 71, 72, 105, 136, 138, 368
82, 10, 62, 72, 74, 369
83, 44, 47, 68, 70, 370
84, 83, 166, 167, 370
85, 152, 370
86, 148, 196, 197, 227, 307, 371
87, 19, 23, 49, 50, 51, 52, 53, 54, 55, 63, 94, 96, 102, 127, 159, 166, 171, 212, 213, 237, 271, 272, 312, 372
88, 373
89, 374
90, 19, 49, 63, 102, 106, 111, 112, 127, 157, 159, 212, 233, 234, 248, 274, 275, 277, 278, 323, 375
91, 19, 49, 102, 127, 233, 274, 275, 323, 376
92, 111, 157, 158, 212, 278, 323, 376
93, 19, 49, 102, 109, 112, 113, 127, 159, 212, 248, 323, 376
94, 108, 183, 186, 210, 246, 249, 250, 252, 255, 377
95, 144, 147, 183, 184, 185, 222, 223, 246, 248, 249, 252, 254, 377
96, 40, 105, 143, 144, 148, 183, 184, 186, 196, 198, 206, 226, 249, 250, 251, 252, 254, 378
97, 379
98, 249, 251, 252, 253, 260, 261, 380
99, 14, 15, 135, 141, 142, 147, 151, 198, 221, 225, 226, 293, 294, 307, 381
100, 99, 124, 125, 130, 186, 188, 221, 222, 228, 382
101, 144, 383
102, 18, 38, 39, 86, 109, 110, 156, 157, 384
103, 150, 181, 203, 308, 385
104, 151, 183, 309, 385
105, 18, 48, 111, 112, 158, 159, 160, 161, 174, 175, 212, 213, 235, 278, 279, 282, 324, 325, 386

Figure 106, 160, 325, 387
107, 161, 325, 387
108, 161, 325, 387
109, 152, 183, 248, 255, 256, 257, 260, 299, 322, 388
110, 152, 255, 256, 259, 260, 299, 322, 389
111, 151, 205, 389
112, 151, 206, 322, 389
113, 206, 322, 389
114, 200, 308, 390
115, 200, 308, 390
116, 181, 200, 308, 390
117, 202, 308, 390
118, 182, 202, 203, 308, 390
119, 203, 390
120, 203, 308, 390
121, 14, 21, 22, 163, 164, 165, 166, 167, 168, 315, 391
122, 264, 269, 316, 392
123, 264, 269, 316, 392
124, 269, 316, 392
125, 263, 264, 269, 316, 318, 392
126, 269, 316, 392
127, 316, 392
128, 163, 164, 181, 265, 266, 267, 315, 317, 393, 394, 395
129, 181, 182, 210, 265, 266, 267, 269, 270, 317, 320, 396
130, 181, 182, 265, 266, 317, 319, 396
131, 182, 265, 267, 270, 317, 319, 397
132, 265, 267, 268, 317, 397
133, 172, 272, 321, 397
134, 172, 193, 272, 321, 398
135, 172, 272, 321, 398
136, 272, 321, 398, 399, 400, 401
137, 193, 236, 273, 321, 401
138, 275, 323, 402
139, 275, 323, 402
140, 19, 127, 218, 274, 275, 276, 277, 323, 402
141, 19, 127, 218, 274, 275, 277, 282, 323, 402
142, 14, 54, 127, 154, 158, 174, 175, 219, 234, 235, 275, 276, 277, 278, 279, 280, 282, 283, 284, 286, 314, 323, 324, 402
143, 14, 127, 174, 234, 275, 277, 278, 279, 280, 284, 314, 323, 326, 402
144, 174, 235, 275, 279, 282, 323, 324, 326, 403
145, 174, 275, 282, 323, 325, 326, 403
146, 234, 296, 323, 403
147, 323, 403
148, 235, 237, 403
149, 13, 14, 19, 54, 174, 274, 403

Figure 150, 174, 175, 403
151, 126, 226, 293, 404
152, 126, 227, 254, 404
153, 126, 222, 223, 294, 404
154, 126, 179, 294, 404
155, 126, 179, 223, 294, 404
156, 126, 224, 294, 404
157, 126, 223, 294, 405
158, 126, 179, 224, 294, 405
159, 126, 224, 294, 405
160, 126, 222, 224, 245, 294, 302, 405
161, 126, 224, 302, 405
162, 126, 179, 224, 225, 245, 294, 302, 406
163, 126, 179, 206, 225, 309, 406
164, 126, 205, 206, 223, 225, 309, 406
165, 126, 179, 197, 227, 293, 295, 406
166, 126, 226, 406
167, 234, 236, 296, 407
168, 20, 52, 133, 134, 193, 237, 238, 296, 407
169, 134, 297, 408
170, 133, 180, 193, 231, 243, 244, 300, 301, 408
171, 133, 203, 205, 244, 301, 302, 322, 408
172, 133, 245, 302, 322, 409
173, 175, 182, 278, 283, 284, 324, 326, 409, 410
174, 127, 133, 150, 193, 210, 225, 230, 241, 294, 299, 310, 411
175, 127, 133, 210, 225, 241, 294, 299, 310, 412
176, 151, 210, 225, 299, 310, 413
177, 210, 225, 299, 310, 414
178, 127, 133, 150, 209, 210, 225, 240, 298, 311, 415
179, 127, 151, 209, 210, 225, 298, 310, 311, 416
180, 133, 193, 225, 228, 239, 240, 297, 417
181, 181, 211, 225, 297, 311, 418
182, 133, 193, 228, 239, 240, 297, 298, 419
183, 133, 180, 193, 225, 239, 240, 297, 298, 420
184, 222, 295, 301, 421
185, 72, 222, 295, 301, 421
186, 224, 225, 245, 295, 301, 302, 421
187, 151, 205, 206, 223, 295, 309, 421
188, 205, 206, 223, 309, 422
189, 245, 319, 320, 422
190, 181, 200, 203, 302, 308, 422
191, 199, 222, 294, 307, 422
192, 205, 422
193, 236, 240, 423

Figure 194, 133, 192, 239, 296, 423
195, 133, 296, 423
196, 133, 179, 180, 192, 193, 296, 423
197, 133, 179, 180, 192, 423
198, 133, 180, 236, 423
199, 133, 236, 242, 423
200, 172, 216, 232, 242, 273, 300, 424
201, 172, 216, 232, 242, 273, 424
202, 133, 179, 180, 193, 243, 300, 301, 424
203, 133, 179, 180, 193, 243, 300, 301, 424
204, 133, 179, 180, 193, 243, 300, 301, 424
205, 133, 179, 180, 193, 244, 300, 301, 302, 425
206, 133, 179, 180, 193, 231, 243, 300, 301, 425
207, 133, 180, 226, 253, 295, 300, 301, 426
208, 199, 294, 307, 426
209, 197, 227, 426
210, 198, 199, 226, 294, 307, 426
211, 202, 308, 427
212, 202, 308, 427
213, 180, 259, 295, 309, 427
214, 259, 299, 309, 322, 428
215, 151, 199, 206, 225, 295, 309, 428
216, 151, 206, 225, 254, 295, 309, 428
217, 251, 253, 254, 309, 323, 429
218, 221, 314, 429
219, 221, 314, 429
220, 153, 181, 215, 216, 313, 429
221, 154, 215, 216, 220, 276, 314, 430
222, 216, 221, 276, 314, 430
223, 430
224, 151, 193, 206, 223, 309, 431
225, 432
226, 287

Fillet, accessory, 64
Fissures, cerebral, 402
spinal, 24
Flaccid paralysis, 179
paraplegia, 301
Flaccidity, 126, 203, 231, 238
Flechsig's order of myelination, 218
oval field, 138
Flexion, rigidity of legs in, 193
Flocculus, 315
Focal symptoms, 323
in calcarine area, 325
in central convolution, 323, 324
in corpus callosum, 326
in frontal convolution, 324
in frontal gyrus, 324
in hippocamal gyrus, 325

Focal symptoms, in insula, 325
in parietal convolution, 324
in pyriform area, 325
in supramarginal convolution, 324
Foot, adduction reflex of Marie and Meige in, 232
perforating ulcer of, 203, 390
Forced rotatory movements, 264
Forel's decussation, 46, 49, 94, 95
Form, center for, 54
sensation of, 305
Formatio reticularis, 28, 33, 62, 77, 81
Fountain decussation, 49
Fourth sensory neuron, 141
Fourth ventricle, 29, 35, 93
Foville's syndrome, 419
Fracture dislocation of cervical vertebræ, 429
Friedreich's ataxia, 205, 245, 319, 320, 422
Frontal convolution, focal symptoms in, 324
Frontal gyrus, focal symptoms in, 324
Frontopontine fibers, 37, 96
tract, 51
Functional paralysis following section of posterior nerve roots, 145
Funiculus cuneatus, 31, 76, 77, 139
gracilis, 31, 76, 77, 78, 138

Gait, cerebellar, 269, 270, 318, 396
hemiplegic, 424
paraplegic, 425
Ganglion or ganglia, 11
basal, 271, 372
habenulæ, 53
infracortical, 12
of cranial nerves, 198, 307
of posterior nerve roots, 147, 198
semilunar, lesions of, 307
Gelatinous substance, 31, 41, 59, 61, 75, 78, 80
Geniculate body, lateral, 48, 52, 96, 160, 212
medial, 47, 52, 91, 96, 97, 98, 212
Genito-urinary center, 247
Glioma, 346, 389, 421
Gliomatosis, 225
Gliosis, of spinal cord, 309, 310
of fourth ventricle, 309
Globus pallidus, 23, 55, 94, 97, 171, 272
atrophy of, 400
progressive degeneration of, 399
Glossopharyngeal nerve, 34, 109, 137, 155
nucleus of, 81, 100, 123
Gluteal reflex, 184
Glycosuria, nucleus for, 210
Golgi cells, type I., 7, 8, 9
type II., 9, 26, 55, 345
Goll, column of, 31
Gombault, sacral triangle of Philippe and, 138
Gray columns, 26, 58, 59, 60, 61
commissure, 24, 27
matter, 60, 61
of medulla, 29
reticulation, 33
Gubler, syndrome of, 420
Gudden's commissure, 158
Gustatory fibers of chorda tympani, 156
Gustatory sense, 109
Gyrus, frontal, focal symptoms in, 324
hippocampal, 113
focal symptoms in, 325

Hair sense, neuron for, 135
test for, 15
Hand, traumatic palsy of, 226
Hardening, 3
Head, afferent paths for, 17
attitude of, in cerebellar lesions, 264
common sensations in, paths for, 17, 19
vegetative innervation of, 433
Head's classification of aphasias, 287
Hearing, physiology of, 158
sense of, neurons for, 109, 110
Heat, neurons for, 103, 104, 151
sensation of, 17, 135, 137, 304
test for, 14, 142
Heidenhain's iron-hematoxylin technique, 11
Hematomyelia, 259, 309, 427
Hemianæsthesia, 430
Hemianopsia, 160, 387
Hemiataxia, 181
Hemiopic pupillary reaction, Wernicke's, 161
Hemiparaplegia, 180
Hemiplegia, 20, 179, 180, 238, 423, and *see* Paralysis
capsular, 429
congenital double, 423
double, 241, 423
Hemiplegic gait, 423
Hemispheres, cerebral, lesions of, 323
localizing areas of, 402
Hemorrhage, capsular, 194, 429
Hereditary cerebellar ataxia, 319
spastic paraplegia, 301
Herpes zoster, 147, 198, 307
Hippocampal gyrus, 113, 325
Hoche, bandalette of, 70, 138
septal fibers of, 70
Homolateral pyramidal fibers, 129
pyramidal supply, 51
Homonymous hemianopsia, 387
Huntington's chorea, 273, 322
Hypæsthesia, 180

Hyperæsthesia, 180, 188, 197
Hyperalgesia, 181
Hypertonicity, 191
Hypertrophic syphilitic pachymeningitis, 421
Hypoglossal nerve, 34, 120
  nucleus of, 81, 100
Hypoplasia of cerebellum, 319
Hypothalamic tegmental region, 342
Hypotonia, 220
  cerebellar, 265, 393
Hysterical paralysis, 303

Ideational agnosia, 283
  apraxia, 284
Images, kinæsthetic, 280
Infantile monoplegia, 236, 423
  paralysis, 126, 404
Infracortical ganglia, 12
  motor mechanism, 261
Inflammation, meningeal, 190
Insula, focal symptoms in, 325
Intercalated cells, 70
Intermediolateral cell column, 60, 76
Internal capsule, 19, 50, 63, 127, 372, and *see* Capsular
  hemorrhage into, 194, 429
  softening of, 237, 238
Interossei muscles, paralysis of, 371
Intracerebellar tumor, 319
Intrapontine course of sixth and seventh nerves, 342
Intrathecal tumors, 190
Iron-hæmatoxylin technique, 11, 56
Irritants and upper motor neuron, 187

Jackson's (Hughling) law, 190
Jacksonian epilepsy, 233
Joint disease, Charcot's, 390
Juxtacerebellar tumors, 321

Kinæsthetic images, 280
Knee jerk, in cerebellar lesions, 318

Lancinating pains in tabes dorsalis, 201
Landry's paralysis, 295
Lateral lemniscus, *see* Lemniscus
Lateral nucleus, 18, 53, 84, 215
Lateral sclerosis, 231, 301, 408
  amyotrophic, 245, 302, 421
  primary, 242
Legs, rigidity of, in flexion, 193
Lemniscus, lateral, 38, 43, 44, 46, 47, 86, 91, 92 93, 95, 98, 110
  medial, 16, 31, 37, 38, 43, 44, 46, 47, 52, 71, 79, 86, 91, 92, 93, 95, 97, 139, 211, 297, 298, 310
Lenticular degeneration, 272, 321, 397, 398
Lenticular lesions, 97, 272
Lenticulostriate artery, 237
Lentiform tone controlling influence, 47
Leprosy, 422
Leptomeninges, sarcomatosis of, 421
Leptomeningitis, syphilitic, 422
Lethargic encephalitis, 94, 274
Lightning pain in tabes dorsalis, 201
Linking of neurons, 14
Lissauer's column, 26, 58
Little's disease, 241
Local spasm, tonic, 187
Localization, 14, 15, 127, 218
Localizing areas, 375, 402, 403
Locus coeruleus, 42
Locomotor ataxia, *see* Tabes dorsalis
Longitudinal bundles, 84, 209
  fasciculi, 33, 39, 44, 46, 47, 80, 82, 92, 97
Lower motor neurons, 12, 20, 62, 64, 66, 99, 100, 101, 119, 120, 125, 186, 344
  convulsions, 187
  higher control of, 124
  paralysis of, 38, 125, 126, 221, 293, 302, 406, 426
Lower motor sensory nuclei, 210
Lower pontine syndrome, 430
Lower somatic neuron, 125
Lower splanchnic neuron, 102, 125
Lowest sensory neuron, 13, 65
Lumbar cord, lesion at, 252
  section through, 56
Lumbar enlargement, 75, 119
Lumbar nerve, 65
Lumbar nerve root, compression of, 367
Lumbar segment, 66, 73, 334, 336, 348
Lumbosacral enlargement, 99
Lumbrical muscles, paralysis of, 371
Luys, nucleus hypothalamicus of, 34

Mammillothalamic bundle, 52
Marchi's technique, 10
Marie and Meige, adduction reflex of, 232
Mastication, muscle sense complex for, 106
Medial lemniscus, *see* Lemniscus
Median nerve, lesions of, 196
  paralysis of, 404
Medulla oblongata, 29, 103, 337
  autonomic control in, 210
  centers in, 33
  central canal of, 29
  cranial nerve nuclei in, 34
  direct pyramidal tract in, 30
  gelatinous substance of, 31
  gray matter of, 29
  lesions in, 241, 279, 310
  longitudinal tracts in, 33

Medulla oblongata, median longitudinal fasciculus of, 33
median raphe of, 34
lower motor neurons in, 100
pyramids of, 30
pyramidal decussation in, 30
pyramidal lesions in, 241, 299
section through, 28, 338, 347, 350, 351, 352, 353, 354, 356
sensory tracts in, 207
superior velum, 43, 356
Meige, foot adduction reflex of Marie and, 232
Meningeal inflammation, 190
Mesencephalic nuclei of fifth nerve, 41
root of fifth nerve, 41, 43, 89, 93
syndrome, 417, 418
tegmental lesion, 311
Mesencephalon, 63, 140, 239, 337
lesions of, 210, 297
paralyses of, 239
section through, 46, 94, 341
tectum of, 94
tegmentum of, 94
Meynert's decussation, 49, 94, 95
Millard Gubler, syndrome of, 420
Mind blindness, 283
deafness, 283
Mixed lesions of motor and sensory tracts, 322
Mixed nerves, 14
lesions of, 196
Monakow's bundle, 95
Monoplegia, 180
cortical, 236
facial, 236
facio-brachial, 423
infantile cerebral, 236, 423
Motion, paralysis of, causes of, 292
passive, sense of, 135
voluntary, 186
Motor apraxia, 283, 284, 409, 410
Motor areas of cortex, 49, 375, 376, 402
Motor loss in lesions of sensory cortex, 220
Motor mechanism, 171, 261
Motor nerve roots, avulsion of, 65
Motor neuron, 60, 119
diseases of, 292
Motor neurons, lower, *see* Lower motor neurons
convulsions, 187
of cranial nerves, 120
higher control of, 124
paralysis of, 38, 125, 126, 221, 245, 293, 302, 406, 426
Motor neurons, upper, *see* Upper motor neurons
Motor and reflex inhibitory, voluntary, 102
Motor and sensory tracts, mixed lesions of, 302, 322
Motor nucleus of trigeminus, 124
Motor paralyses, 179, 221, 292
Motor sensory nuclei, 210
Motor tracts in brain and cord, 19
Movements, athetoid, 216
forced rotatory, 264
sense of, 305
skilled, 229
Multiple sclerosis, 245, 302
Muscles, segmental supply of, 330, 331
Muscle sense, complex, 106
paths for, for fifth nerve, 139
Muscle tone, 190
loss of, 145, 203
nervous influences underlying, 382
Muscle-tendon-joint sense, 15, 16, 105, 106, 135, 138, 143, 150
Muscular atrophy, 146, 405, 406
Muscular lesions, paralysis due to, 221, 293
Musculospiral nerve, lesions of, 196
paralysis of, 226, 404
Myelin sheath, 6
Myelinated cutaneous nerves, 14
Myelination, Flechsig's order of, 218
Myelination of fibers, in pons, 37

Neostriatum, 23, 24, 55, 172
Nerve cells, 5, 6, and *see* Cells
tigroid substance of, 11
Nerve roots, anterior, 57
avulsion of, 65
dorsal, 363
dermatoses corresponding to, 378
skin areas corresponding to, 378
posterior, 57, 199
lesions of, 307
section of, 66, 144, 145, 146
Nerve tracts, 14, 361
anatomy of, 3, 117
degeneration of, 62, 346
lesions of, 303, 196
physiology of, 117
Nerve, or Nerves
abducent, 39, 89, 342
lesions of, 311
nucleus of, 39, 87, 89, 100, 103, 120
accessory, 34, 78, 120, 122
nucleus of, 62
auditory, 23, 35, 109
cervical, 78, 88, 333
chorda tympani, 109
cochlear, 38, 85, 109, 156, 166, 384
nucleus of, 38, 85
cranial, and *see* Nerves, first, second, etc.

Nerve, cranial, anterior roots of, 225
lower motor neurons of, 120
motor roots of, 225
nerve roots of, 210
nuclei of, 34, 337, 338
superficial origin of, 336
eighth, 23, 35, 109
eleventh, 34, 78, 120, 122
nucleus of, 62
facial, 90, 108, 123, 342
ganglia of, 198
lesions of, 311
nucleus of, 39, 90, 100, 103, 123
fifth, 40, 78, 79, 83, 87, 91, 104, 106, 108
ganglia on, 198, 307
lesions of, 298, 307, 311
mesencephalic root of, 41, 43, 89, 93
nuclei of, 40, 41, 88, 100, 103, 124, 139
spinal tract of, 31, 40, 61
first, 162
fourth, 91, 92, 121
decussation of, 44, 92
nucleus of, 44, 93, 101, 102
glossopharyngeal, 34, 109, 137, 155
nucleus of, 81, 100, 123
hypoglossal, 34, 120
nucleus of, 81, 100
lumbar, 65
median, lesion of, 196
paralysis of, 404
mixed, 14
lesions of, 196
musculospiral nerve, paralysis of, 226, 404
myelinated, cutaneous, 14
ninth, 34, 109, 137, 155
nucleus of, 81, 100, 123
oculomotor, 95, 98, 121
lesions of, 297
nucleus of, 47, 96, 98, 101, 102
olfactory, 162
optic, 111, 166
lesions of, 386
projection path for, 386
peripheral, lesions of, 148, 196, 307
penumogastric, *see* Nerve, vagus
radial, lesions of, 196
paralysis of, 226, 404
sciatic, 346
second, 111, 166
lesions of, 386
projection paths for, 386
sensory, 14, 141, 303
interruption of, 197
lesions of, 196, 303
plexus, 347
synapses, 346
seventh, 90, 108, 123, 342
Nerve, seventh, ganglia of, 198
lesions of, 311
nucleus of, 39, 90, 100, 103, 123
sixth, 39, 89, 342
lesions of, 311
nucleus of, 39, 87, 89, 100, 103, 120
special sense, 154
spinal, 361, 381
lesion of, 226
rami of, 147, 198
visceral sense in, 108
summary of, 117
tenth, 34, 109, 155
ganglia of, 198
nucleus of, 81, 100, 123
third, 95, 98, 121
lesions of, 297
nucleus of, 47, 96, 98, 101, 102
trigeminus, 40, 78, 79, 83, 87, 91, 104, 106, 108
ganglia on, 198, 307
lesions of, 298, 307, 311
mesencephalic roots of, 41, 43, 89, 93
nuclei of, 40, 41, 88, 100, 103, 124, 139
spinal tract of, 31, 40, 61
trochlear, 91, 92, 121
decussation of, 44, 92
nucleus of, 44, 93, 101, 102
twelfth, 34, 120
nucleus of, 81, 100
ulnar, lesion of, 196
paralysis of, 227, 404
section of, 148, 371
unmyelinated cutaneous, 15
vagus, 34, 109, 155
ganglion of, 198
nucleus of, 81, 100, 123
vestibular, 35, 41, 69, 82, 84, 109, 163, 164, 393
Nervous diseases, classification of, 292
symptoms of, 186
Nervous system, 5, 362
Nervus intermedius, 90
Neurilemma, 6
Neuritis, 293
peripheral, 197, 227, 406
Neuroglia, 5, 57
Neurons, 6
afferent, 8, 13
association, 13, 67
centripetal, 8
cerebellar, 14, 166
classification of, 12
cortico-ponto-cerebellar, 166
descending association, 67
diseases of, 186, 292
efferent, 12, 102, 117, 125

Neurons, endogenous, 66, 67
for cold, 103, 104
for heat, 103, 104
for muscle-tendon-joint sense complex, 106
for pain, 103, 104
linking of, 14
lower motor, *see* Lower motor neurons
motor, *see* Motor neurons
peripheral, 8
pyramidal, 12, 228
relays, 13
retinal, 111
sensory, 8, 13, 88, 103, 104, 108, 112, 134, 137, 140, 141, 152, 153, 188, 307
somatic efferent, 117, 125
spinal, 13, 66, 67
splanchnic, 117, 125
summary of, 99
thalamocortical, 13, 106, 141, 153
tract forming, 66
upper motor, *see* Upper motor neurons
Neurology, applied, 179
Nissl bodies, 345
Nominal aphasia, 288
Nucleus, 11
ambiguus, 34, 81, 100, 122
arcuate, 34
auditory, 157
caudate, 55, 272
celebellar, 42
cochlear, 38, 85
cuneatus, 31, 71, 72, 79, 139
Deiters', 23, 36, 42, 68, 69, 84, 164, 209, 352, 364
dentatus, 42, 84, 340
dorsalis, 21, 27, 60, 76, 215
emboliformis, 42
fastigii, 42, 84, 85
globosus, 42, 85
glycosuria, for, 210
gracilis, 31, 71, 72, 78, 79, 138, 139
hypothalamicus of Luys, 23
lateralis, 32
of Bechterew, 163
of lateral lemniscus, 44
of Stilling, 27
parasympathetic, 210
pontis, 37, 85
red, 23, 46, 47, 52, 96, 97, 211, 321
roof, 84, 85
sensory, 18, 210
splanchnic, 100
tecti, 42, 85
terminal, 12
thalami, 41, 53, 213, 312
ventrolateral, 215
Nystagmus, 263
Nystagmus in cerebellar lesions, 318
late, 270

Oculomotor nerve, 95, 98, 121
lesions of, 297
nucleus of, 47, 96, 98, 101, 102
paralysis, 225
Olfactory area, 162
nerve, 162
paths, 112
tract, lesions of, 162
Olive, accessory, 32, 83
inferior, 32, 79, 80, 82, 167, 348, 351, 370
superior, 39, 86, 89, 110
Olivocerebellar bundle, 167
connections, 82
fibers, 83, 352
Optic fibers, 160
nerve, 111, 166
lesions of, 386
projection paths for, 386
radiation, 98, 112, 212
tract, 48, 96, 97, 98, 160, 212, 343, 360
Orientation, in space, 164
Oval field, 76, 138

Pachymeningitis, hypertrophic syphilitic, 421
Pain, deep, test for, 15
fibers for, 137
intense, 189
lancinating, 201
lightning, 201
neurons for, 103, 104, 135, 136, 151
paroxysmal, 189
pressure, 107
referred, 186
rheumatic, 189
sensation of, 17, 135, 142, 304
skin, 15
thalamic, 215
Painful stump, 197
Pal-Weigert technique, 11
Palæostriatum, 23, 24, 54, 171, 271
Pallidal system, degeneration of, 401
Palsy, birth, 406
traumatic, of hand, 226
Paræsthesia, 181, 188
Paralysis agitans, 94, 273, 321, 401
Paralysis, 179
arteries involved in, 237
bilateral, 300
Brown-Sequard, 107, 152, 255, 258, 322, 388, 389
bulbar, 230, 245
capsular, 237
causes of, 292
cortical, 235, 283

Paralysis, crossed, 180, 298
distribution of, 228
due to muscular lesions, 221, 293
familial periodic, 303
flaccid, 179
functional, 145
hysterical, 303
infantile, 126, 404
Landry's, 295
mesencephalic, 239
motor, 179, 221, 292
oculomotor, 225
periodic, 303
pontine, 239
posture in, 192
of interossei muscles, 371
of lower motor neuron, *see* Lower motor neuron
of lumbrical muscles, 371
of median nerve, 404
of musculospiral nerve, 226, 404
of radial nerve, 226, 404
of ulnar nerve, 227, 404
of upper motor neuron, *see* Upper motor neuron
reflex, 303
sensory, 179, 283
spastic, 179, 192, 243
without known lesions, 303
Paralytic stroke, 237
Paraplegia, 180, 242, 300, 424
Erb's syphilitic, 244
flaccid, 301
hereditary, 301
rigidity of, 193
spastic, 179, 243, 301, 424, 425
syphilitic, 244, 425
Paraplegic gait, 424
Parasympathetic nuclei, 210
Paresis, 179
in cerebellar lesions, 265
Parietal convolution, focal symptoms in, 324
Parietal cortex, localization of sensation in, 218
relay from thalamic nucleus to, 213
Parietal lesions, sensory loss in, 220
Parkinson's disease, 321
Pars basilaris pontis, 36, 85, 297, 298, 320
Pars tegmentalis pontis, 36, 38, 86, 87
Passive motion, sense of, 135
position, sense of, 135
Peduncles, 37, 45, 212, 297
Pelvic cancer, 152
Perforating ulcer of foot, 203, 390
Perineum, sensory areas in, 379
Peripheral muscles, paths from to vermis, 165
Peripheral nerves, distribution of, 143
lesions of, 148, 196, 307
Peripheral neuritis, 197, 227
alcoholic, 406
Peripheral polyneuritis, 295
Perseveration, 284
Phenomena, concomitant, 232
rebound, 268
Philippe, sacral triangle of, 70
Pia, 57
Plantar reflex, 184
Plexus, sensory nerve, 347
Pneumogastric nerve, *see* Nerve, vagus
Pointing test of Barany, 269
Poliomyelencephalitis, 404
Poliomyelitis, 223, 365, 404, 405
acute anterior, 67, 126, 223
anterior, 64
chronic anterior, 126
Polyneuritis, peripheral, 295
postinfluenzal, 426
Pons, 103, 337
autonomic control in, 210
basilar portion of, 36, 85, 297, 298, 320
lesions in, 240, 298, 310
lower motor neurons in, 100
medial longitudinal fasciculus of, 39
myelination of fibers in, 37
nuclei of, 37, 85
of fœtus, 337
section through, 36, 42, 85, 93, 339, 340, 353, 354, 355, 356, 357, 358
sensory tracts in, 207
striæ medullares of, 38
tegmental part of, 36, 38, 86, 87
upper end of, 43
Pontine lesions, 240, 298, 310
paralysis, 239
syndrome, 415, 416, 420
Porencephaly, double, 237
Position of limb, sense of, 305
Posterior radiculitis, 307
rami of spinal nerves, 147
root ganglia, 147, 198
sclerosis, 149
spino-cerebellar tract, 21, 60, 76, 83
spinothalamic tract, 104, 136, 151, 154, 208, 385
Posterolateral column, 58, 61, 75
fasciculus, 61
sclerosis, 302
Postinfluenzal polyneuritis, 426
Posture, center for, 54
Posthemiplegic athetosis, 421
Pre-ataxic tabes, 427
Prefrontal area, 279
Prepyramidal tract, 95

Pressure pain, 107, 135
Pressure sense, 15, 17, 107, 135, 139, 143, 152, 305
Primary lateral sclerosis, 242
  posterior sclerosis, 203, 308
Processes of nerve cells, 6
Progressive muscular atrophy, 405, 406
Prosencephalon, ventral aspect of, 343
Protopathic sense, 15, 135, 142
Pseudorigidity, 405
Pulvinar, 48, 52, 160, 212
  section through, 360
Pupillary reaction, hemiopic, of Wernicke, 161
Purkinje axons, 167, 168
Purkinje cells, 8, 37, 167, 168, 345
Pyramids, 30, 64, 80, 93
Pyramidal axons, disease near, 188
  cells, 188, 344, 345
  control, loss of, 192
  decussation, 30, 62, 64, 78, 79, 99, 103, 230, 350, 351
  fibers, 37, 43, 45, 46, 50, 64, 85, 91, 93, 94, 127, 129
  lesions, 130, 192, 228, 229, 231, 232, 233, 235, 239, 241, 297, 298, 299
  neuron, 12, 228
  supply, homolateral, 51
  system, 130, 228, 229
  tract, 19, 20, 30, 91, 96, 128, 133, 166, 208
Pyriform area, focal symptoms in, 325

Quadrantal hemianopsia, 387

Radial nerve, lesion of, 196
  paralysis of, 226, 404
Radiation, auditory, 157, 158
  thalamic, 54
Radicular areas, 198
Radiculitis, anterior, 225
  posterior, 189, 199, 225, 307
  syphilitic, 199, 225, 426
Rami, of spinal nerves, 147
Rami communicantes, 247
Reaction, Wernicke's hemiopic pupillary, 161
Rebound phenomenon, 268
Receptive end organs, 8, 9
Rectoanal center, 247
Red nucleus, 23, 46, 47, 52, 96, 97, 168, 211, 321
Referred pain, 186
Reflex, adduction, 232
  arc, 118, 302, 363
  bulbocavernous, 184
  centers, 246, 248, 377
Reflex, cremasteric, 184
  cutaneous, 184, 185, 194
  defense, 193
  epigastric, 184
  foot adduction, of Marie and Meige, 232
  gluteal, 184
  in cerebellar lesions, 270
  in pyramidal lesions, 231
  in spinal cord lesions, 193
  in tabes dorsalis, 202
  inhibitory, 102
  paralysis, 303
  plantar, 184
  scratch, 13, 363
  stem, 173
  submammary, 184
  subumbilical, 184
  supraumbilical, 184
  tendon, 185, 232
  thumb, 232
  visual, tract, 112
Reflexes, 186
  mechanism of, 13
Relay, from dorsolateral thalamic nucleus to parietal cortex, 213
  to essential thalamic organ, 213
Restiform body, 31, 35, 82, 83, 210, 338
  lesions of, 320, 396
  section through, 74
Reticularis alba, 33, 82
  grisea, 33, 82
Reticulation, gray, 33, 82
  white, 33, 82
Retina, 111
Retinal neurons, 111
Retrograde degeneration, 11
Retro-olivary bulbar syndrome, 412, 413, 414
Rheumatic pains, 189
Ribs, cervical, 426
Rigidity, 321
  decerebrate, 190, 290
  in upper motor neuron lesions, 231
  of legs, in flexion, 193
  of paraplegia, 193
Rolando, substantia gelatinosa of, 26
Romberg's sign, 202
Roof nuclei, 84, 85
Rotatory movements, forced, 264
Roughness, sense of, 305
Rubrospinal decussation, 46, 49, 94, 95, 359
Rubrospinal tract, 22, 33, 44, 47, 68, 80, 83, 92, 93, 96, 208
  degeneration of, 370
  lesions of, 297, 311
Rubrothalamic fibers, 96

Sacral segment, 66
lesion of, 253
section through, 67, 335, 336
Sacral triangle, 66, 70, 138
Sarcomatosis of leptomeninges, 421
Sciatic nerve, 346
Sclerosis, amyotrophic lateral, 245, 302, 421
cerebrospinal, 302
combined, 205, 244, 302, 408
disseminated, 321
lateral, 231, 242, 245, 301, 302, 408, 421
multiple, 245, 302
posterior, 149, 203, 308, 385
posterolateral, 302
primary, 203, 308, 385
syphilitic, 301
Scratch reflex, 13, 363
Section cutting, 4
Segmental areas, 198, 383
relations of spinal cord, 183, 380
topography of, in spinal cord, 380
Segmental supply of muscles, 330, 331
Semantic aphasia, 288
Semilunar ganglion, lesion of, 307
Sensation, common, 17, 19
localization of in parietal cortex, 218
Sense, auditory, 18
body, 14, 217
bone, 15
epicritic, 135, 142
gustatory, 109
of cold, 142
of hearing, 109, 110
of heat, 142
of movement, 305
of position of limbs, 305
of roughness, 305
of sight, 18
of size, 305
of texture, 306
of weight, 135
pressure, 17, 107, 135
protopathic, 15, 135, 142
somatic, 14
stereognostic, 135
tactile, 17, 135, 142
tuning fork, 143
vestibular, 18
vibratory, 107, 135, 143
visceral, 108, 154
Senses, loss of in thalamic lesions, 313
Sensibility, deep, 15, 135, 143
Sensory areas, 183, 190, 217, 218, 219, 220 314, 375, 376, 379, 402
aura, 234
Sensory areas, conducting paths, lesions in, 306
cortex, motor loss in lesions of, 220
disturbances of reflex arc, 302
loss, in capsular lesions, 221
in subcortical parietal lesions, 220
nerves, 14
distribution of peripheral, 143
lesions of, 196, 197, 303
physiology of, 141
plexus, 347
synapses, 346
neurons, 8, 13, 88, 103, 104, 108, 112, 134, 137, 140, 141, 152, 153, 188, 307
paralysis, 179, 283
tracts, 14, 16, 207
lesions of, 196, 302, 303, 308
paralysis of, 302
physiology of, 141
Septal fibers, of Hoche, 70
Septomarginal bundle, 66, 70, 138
fasciculus, 76
Shock, 194
Sight, sense of, 18
Silent areas of cortex, 174, 326
Size, sense of, 305
Skew deviation of eyes, 18, 263
Skilled movements, 229
Skin areas, sensory, 183
corresponding to dorsal nerve roots, 378
Skin pain, test for, 15
Smell, center for, 282
Softening, of brain, 127, 366
of cerebellum, 320
of internal capsule, 237, 238
subcortical, 236
Somatic sense, 14
Space, Virchow-Robin, 5
Spasm, torsion, 401
Spastic diplegia, 241, 426
paralysis, 179, 192, 243
paraplegia, 179, 243, 301, 424, 425
Spasticity in upper motor neuron lesions, 231
in spinal cord lesions, 193
Special sense, nerves of, 154
Speech, 235, 279
in cerebellar lesions, 270, 318
Spinal column, carcinoma of, 64
Spinal cord, association neurons in, 13
central canal of, 27
crush of, 253
degeneration of, 69, 70, 72, 368, 369
efferent paths from, 20
endogenous fibers of, 365
fasciculi proprii of, 13
fibræ propriæ of, 173

Spinal cord, fœtal, 74
formatio reticularis of, 28
gliosis of, 309, 310
gray commissure of, 24, 26, 27
internal anatomy of, 3
lesions of, 69, 72, 149, 193, 198, 230, 245, 248, 249, 250, 251, 252, 253, 255, 259, 299, 368, 428, 431
microscopical study of, 56
motor tracts in, 19
nerves of, 361
nucleus dorsalis of, 27
reflex centers in, 246, 248, 377
reflex mechanism in, 13
section of, 24, 56, 363
segmental relations of, 183, 380
topography of, 380
sensory tract in, 16
Spinal nerves, 147, 381
lesion of, 226
visceral sense in, 108, 154
Spinal vessels, 28
Spinocerebellar ataxia, 319, 320
Spinocerebellar tracts, 21, 31, 32, 43, 60, 68, 71, 76, 77, 78, 80, 83, 87, 165
Spino-olivary fasciculus, 68, 167
tract, 83
Spinotectal tracts, 33
Spinothalamic tracts, 33, 80, 92, 104, 136, 138, 151, 154, 207, 208, 309, 311, 370, 385
Splanchnic efferents to third nerve, 47
Spontaneous deviation of arm, 268
Standing, in cerebellar lesions, 269
Stem reflexes, 173
Stereognostic sense, 135
Stilling, nucleus of, 27
Stratum profundum, 48
zonale, 97
Striæ medullares, of pons, 38
Striatal degeneration, 401
Striate body, and its connections, 23
Stroke, paralytic, 237
Stump, painful, 197
Subcortical apraxia, 284
Subcortical lesions, 235, 296
parietal, 220
pyramidal, 235
Subcortical softening, 236
Submammary reflex, 184
Substance, tigroid, 11, 345
Substantia gelatinosa of Rolando, 26
nigra, 45, 52, 94, 96
Subthalamic region, 140
sensory lesions of, 211
Subumbilical reflex, 184
Supramarginal convolution, focal symptoms in, 324
Supraumbilical reflex, 184
Sweating, center for, 246
Sydenham's chorea, 274
Synapse, 8, 346
Syndrome, Benedict's, 418
Brown-Sequard's, 230, 428
bulbar, 411, 412, 413, 414
Déjerine's, 313
Foville's, 419
mesencephalic, 417, 418
Millard-Gubler's, 420
pontine, 415, 416, 419, 420
tegmental, 418
thalamic, 215, 313, 429
Weber's, 417
Synergy, 182
Syntactic aphasia, 228
Syphilitic leptomeningitis, 422
pachymeningitis, 421
paraplegia, 244, 425
radiculitis, 199, 225, 426
sclerosis, 301
Syringobulbia, 206, 309
Syringomyelia, 151, 205, 295, 309, 389, 406, 421, 422, 428

Tabes dorsalis, 147, 199, 302, 308, 390, 422, 427
anæsthesia in, 201
ataxia in, 202
crises in, 189
degeneration in, 203
muscle tone in, 203
pain in, 201
reflexes in, 202
trophic disturbances in, 203
Tabetic crises, 189
Tactile discrimination, 14, 106, 304
sense, 17, 135, 142
Tæniæ, 35
Taste, 109, 155, 156
Tear secretion, center for, 246
Tectospinal tract, 23, 33, 44, 68, 80, 82, 92, 93, 97
decussation, 49, 94, 95, 359
Tectum, 94, and *see* Tegmentum
Tegmentum, 94, 342
lesions of, 297, 298, 310, 311
mesencephali, 52, 94
pontis, 36, 38, 86, 87
section through, 342
Tegmental syndrome, 418
tract, 83, 95, 97, 167
Telodendria, 8

Temperature sense, neurons for, 135, 136
test for, 142
Temporal lobe, focal symptoms in, 325
Temporopontine fibers, 37, 51, 96
Tendon reflexes, 185, 232
Texture, sense of, 306
Thalamus, 52, 53, 96, 141, 372
lesions of, 153, 212, 214, 312, 313, 429
nucleus of, 41, 52, 53, 213, 312
Thalamic organ, essential, 19, 53, 140, 213
Thalamic pain, 215
radiation, 54
syndrome, 215, 313, 429
Thalamocortical fibers, 53
neuron, 13, 106, 141, 153
Thermoanæsthesia, 180, 206
Thoracic region, of cord, 65, 69, 72, 73, 119
section through, 59, 67, 251, 252, 334, 335, 348
Thrombosis, of cerebral artery, 234
of spinal artery, 411
of vertebral artery, 412
Thumb reflex, 232
Tickling, 15
Tigroid substance, 11, 345
Tone, 186
Tone controlling influence, 47
Tongue, muscle sense complex for, 106
Tonic convulsions, 187
Tonic mechanism, 21, 23
Tonic spasm, 187
Topognosis, 217
Torsion spasm, 401
Touch, 14, 107, 135, 137, 139, 142, 152
center for, 54
Tracts, summary of, 117
Tractus solitarius, 81
Trapezoid body, 37, 38
Tremor, 321
cerebellar, 267, 397
Triangle, sacral, 66, 70, 138
Trigeminothalamic tract, 18, 31, 33, 80, 87, 88, 89, 92, 104, 137, 208
Trigeminus nerve, 40, 78, 79, 83, 87, 91, 104, 106, 108
ganglia on, 198, 307
lesions of, 298, 307, 311
mesencephalic root of, 41, 43, 89, 93
nuclei of, 40, 41, 88, 100, 103, 124, 139
spinal tract of, 31, 40, 61
Trochlear nerve, 91, 92, 121
decussation of, 44, 92
nucleus of, 44, 93, 101, 102
Trophic disturbances in tabes dorsalis, 203
Trophic lesions after section of posterior nerve roots, 146
Trunk, attitude of in cerebellar lesions, 264
Tubercles in spinothalamic tract, 385
Tuberculum cinereum, 80
cuneatum, 31
Tumor, bulbopontine, 430
intracerebellar, 319
intrathecal, 190
of cerebellum, 319
of dura, 72
Tuning fork sense, 16, 143
vibration, 15, 306
Twitchings, fibrillar, 187

Ulnar nerve, lesion of, 196
paralysis of, 227, 404
section of, 148, 371
Unmyelinated nerve fibers, 7, 15
Upper motor neurons, 12, 50, 63, 102, 227
function of, 130
irritants and, 187
lesions of, 133, 187, 231, 245, 407, 408, 409
paralysis of, 38, 194, 227, 242, 295, 300, 302, 407, 408, 409
physiology of, 127

Vagus nerve, 34, 109, 155
ganglion of, 198
nucleus of, 81, 100, 123
Vegetative innervation of head to face, 433
Velum, superior medullary, 43, 356
Ventricle, fourth, 29, 35, 93
gliosis of, 309
Ventrolateral nucleus, 215
Verbal aphasia, 287, 288
Vermis, 163
afferent tracts to, 165, 315
efferent tracts from, 167, 315
lesions of, 271
Vertebræ, fracture dislocation of, 429
Vertigo, 264
Vestibular apparatus, 163, 164
ataxia, 181
lesions, 392
mechanism, 23
nerve, 35, 41, 69, 82, 84, 109, 163, 164, 393
pathway, 164, 391
sense, 18
Vestibulospinal tract, 23, 33, 36, 69, 84, 164, 209, 311
Vibration, tuning fork, 15, 306
Vibratory sense, 107, 135, 143
Visceral efferents to third nerve, 47
Visceral sense, 108, 154
Virchow-Robin space, 5
Visual field, 160, 387
paths, 21, 158

Visual reflex tract, 112
Vision, physiology of, 159
  tracts for, 111, 112

Wallerian degeneration, 10, 146, 346
Weber's syndrome, 417
Weigert technique, 56
Weight, sense of, 135, 305
Wernicke's hemiopic pupillary reaction, 161
White columns, 57, 58, 60
  commissure, 58
  reticulation, 33, 82
Word blindness, 162
Wrist, drop, 404

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on or before the date last stamped below.

DEC 31 '27
FEB 27 28
FEB 24 29
OCT 30 29
MAR 6 30
MAY 27 30
Aug 8
JAN 18 193
APR 27 1831
OCT 29 1931
FEB 18 1932
FEB 25 1932
APR 26 1932
NOV -9 1932
DEC 15 1932
Feb 6
May 2 33
JUN 17 1933
NOV 15 1933
JUN 16 1934
NOV 25 1934
JUN -7 1935
SEP -4 1936
OCT 28 1937
SEP 12 1938
MAY 15 1941
JAN 13 1943
SEP 20 1949
SEP 20 1949
MAY 13 1950
JAN 22 1983
Feb 5 '83
March '83
MAY 10 2002
APR 22 2002

L341 Keiller, W. 6690
K27 Nerve tracts of
1927 brain and cord.

NAME

J. Thomas
R. R. 084
L. P. Jones
A. Davis
Anderson
K. T.

66900

1941